"*I would have everie man write what he knowes and no more.*"—MONTAIGNE

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EDITORIAL

THE rapid development of methods of anaesthesia in recent years has made it essential for anaesthetists to assume greater responsibilities. Nowadays physicians are rarely sufficiently acquainted with the hazards of modern drugs, or with the techniques of their administration, to make a reliable assessment of a patient's fitness for operation. To avoid the situation where anaesthetists, knowing little of medicine, seek advice from physicians, knowing little of anaesthesia, the anaesthetist's training must be sufficiently broad to enable him to understand, not only the pharmacology of modern anaesthetic agents, but also the additional risks which some patients place on the anaesthetic.

Anaesthetists come into contact with specialists from several branches of medicine and surgery and there is a gap in knowledge which is sometimes difficult to bridge from either side. How much ought the general physician, the cardiologist or surgeon to know about anaesthesia? The rapid progress within the science of healing makes the answer elusive. At this stage specialist groups might with advantage pool some of their resources and take stock.

Within this issue of the Journal modern electrocardiographic theory and techniques are described by Dr. Suffern, a physician, and Dr. Johnstone, an anaesthetist. From Dr. Suffern's account it is apparent that electrocardiography plays such an important part in modern cardiological diagnosis that a complete assessment of the cardiovascular system may be impossible without some knowledge of this subject. Dr. Johnstone describes his use of the electrocardiograph in observing changes in the cardiovascular system during anaesthesia, and states that he believes that this technique provides a valuable objective measure of cardiovascular function. While it is possible to support this view and expect that electrocardiography will be a valuable technique in anaesthetic research, one can only hope that the day will never dawn when continuous electrocardiographic records become obligatory during anaesthesia.

Although recent advances have imposed a a burden of new knowledge on the anaesthetist, they have relieved him of a greater burden of ignorance. Some of the worrying bad risk patients have become fewer and many patients with heart disease can now tolerate major surgery well.

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THE MOVEMENTS OF THE LUNGS

BY

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THE purpose of breathing is to ventilate the alveoli efficiently, but this cannot be accomplished unless the rate and depth of respiration are variable within wide limits. In normal lungs the limits between which the minute ventilation can vary are very wide, but in certain diseases the maximum voluntary minute ventilation may be grossly reduced.

This paper is a brief commentary on some of the results which have been obtained from studies on the mechanical properties of the lungs. The references quoted are to a few papers which illustrate the physiological principles discussed.

FACTORS PRODUCING MOVEMENT OF THE LUNGS

At the end of a normal expiration the lungs are in a state of partial inflation. They are held in this condition as the result of the operation of two opposing forces, namely, the elastic recoil of the lungs tending to collapse them and the resistance of the chest wall opposing further passive movement. The elastic recoil of the lungs can readily be shown to exist by the introduction of air into the pleural space when the lung parenchyma shrinks away from the chest wall.

During inspiration, an outwardly directed force is applied to the lung surfaces. This overcomes the elastic and viscous forces (cf. below) opposing expansion of the lungs, and inspiration occurs. The inflating force applied to the pleura can be measured directly in the pleural space or by measuring the intra-oesophageal pressure (Dornhorst and Leathart, 1952), which is a measure of the intrapleural pressure. Expiration is a passive process.

Campbell (1955) made an electromyographic study of the behaviour of the intercostal muscles in man during quiet breathing and during slight hyperventilation. The lower intercostal muscles contracted during inspiration, but were relaxed during expiration in quiet breathing as well as during hyperpnoea. The evidence concerning the activity of the upper intercostal muscles was not clear, probably because there was considerable technical difficulty in obtaining adequate records from them. Campbell and Green (1955) have studied, similarly, the behaviour of the abdominal muscles. The pattern in this case was complex as it was related to posture as well as to the phases of respiration. In general the muscles were less active during inspiration than during expiration, and, the greater the minute ventilation, the greater was this difference in activity.

FACTORS OPPOSING MOVEMENT OF THE LUNGS

To understand the nature of the factors which oppose inspiration the model described by Christie (1953) is of value (fig. 1). When the bellows are expanded (inspiration) the spring is

FIG. 1

stretched and work is needed to accomplish this. This is exactly analogous to the work required during inspiration to overcome the elastic properties of the lung. In the model the material of which the bellows are made resists movement because of its viscosity. This has its counterpart in the viscosity of the lung tissue. During inflation of the bellows air flows through the nozzle and its flow is resisted, as is the flow of all fluids through tubes. This resistance is analogous to that which hinders the passage of air along the airways of the lungs.

During expiration the lungs become progressively less distended and so the elastic work done during inspiration becomes available to assist expiration. The viscous resistance of the lung tissue to deflation and the viscous resistance due to airflow through the lung airways operate as in inspiration.

Otis, Fenn and Rahn (1950) have shown that when a subject is ventilated artificially in Drinker's machine, the work involved in moving the lungs and chest wall is about 0.4 kg.m per minute. Of this, about 60 per cent is expended in overcoming the elastic resistance of the lung and chest wall and 40 per cent in overcoming viscous resistances. Marshall, McIlroy and Christie (1954) have shown that the work of moving the lungs *alone* is about 0.3 kg.m per minute. Of this, 70 per cent is used in overcoming the elastic resistance of the lung and 30 per cent in overcoming viscous resistances.

The laws governing the flow of gases or fluids through tubes have been known for many years. For any given pressure difference between the ends of a tube the volume of air which passes through the tube in one second depends upon its length and diameter. By halving the diameter the volume of flow per second is reduced to one-sixteenth, whereas, by doubling the length, the volume of flow per second is only halved. These statements are true only under certain conditions of rate of flow and tube geometry which result in the flow being streamlined or laminar. The movement of the gas through the tube under these conditions may be regarded as the telescopic sliding of adjacent concentric layers of gas over one another without mixing. In any one tube, laminar flow is more likely to occur when the velocity of the gas is slow. When the rate of airflow is increased, a stage is reached at which the streamlined flow becomes unstable, and, at even faster rates of flow, turbulent. In streamlined and in turbulent flow two different mathematical expressions relate flow to pressure and tube geometry.

All this basic information has been obtained in laboratory experiments made under conditions of constant flow through smooth straight tubes. The question arises as to whether these physical laws, determined under carefully controlled conditions, can be applied to the airflow through the respiratory passages. During breathing the rate of airflow changes from moment to moment, and the inside of the bronchi and trachea cannot be regarded as smooth. Besides, the air passages branch and alter in diameter in a more or less unknown manner. It is true that a great deal is known about the branchings of the larger airways, but no details could ever become available about the narrower tubes. We are justified in assuming, however, that the physics of gas flow through the air passages of the lungs must be very complex and that the flow of gas is resisted by the narrowness of the tubes.

The work done in moving the lungs during each respiratory cycle is the direct result of the viscous hindrance offered by the tissue of the lungs to inflation and deflation and by the flow of gas along the airways. If the lungs and airways offered no viscous hindrance to inflation and deflation, but were merely perfectly elastic, the simultaneous measurement of intra-oesophageal pressure (which is equivalent to intra-pleural pressure) and tidal volume would show that the most " negative " intra-oesophageal pressure was observed at the summit of inspiration and the least " negative " pressure at the end of expiration. Such, however, is not the case. Figure 2 shows that the intra-oesophageal pressure and tidal volume are a little out of step. The greater this distance, the greater is the amount of work performed during a respiratory cycle.

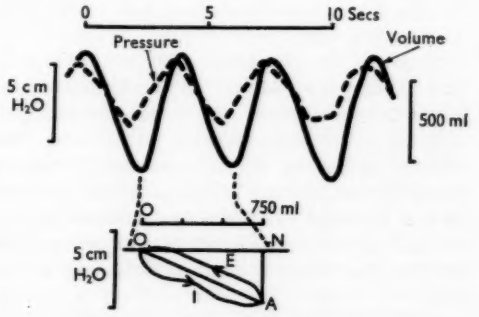

FIG. 2

Simultaneous tracings of intra-oesophageal pressure and tidal volume (above) and the pressure-volume diagram corresponding to one breath (Modified from Marshall et al. (1954).)

Simultaneous tracings of pressure and volume (fig. 2) enable a pressure-volume diagram to be constructed. This relates the changes in intra-oesophageal pressure to the simultaneous change in the volume inspired or expired. The diagram resembles an ellipse. Had the lungs been perfectly elastic and without viscous hindrance, the pressure-volume diagram would have been represented by the major axis of the ellipse. As things are, during inspiration, work (i.e. pressure × volume) is required to distend the elastic lungs. The amount of "elastic work" done in inspiration is represented by the area of the triangle OAN. In addition to the "elastic work" done in inspiration, work is done against viscous resistances and this is represented by the area OIA.

During expiration the energy stored in the lungs as a result of the work done during inspiration against elastic resistance is liberated and is more than sufficient to overcome the viscous hindrance to expiration. The work done against the viscous hindrance to expiration is given by the area OEA, which is clearly less than the "elastic work" (area OAN) recovered from the deflating lungs.

The total work done in one respiratory cycle against viscous resistances is therefore represented by the area OIAE.

The changes in the amount of work necessary to move the lungs have been studied in a number of conditions. Marshall, McIlroy and Christie (1954) and McIlroy and Christie (1954), for example, have applied this method to the study of patients with mitral stenosis and emphysema. In patients with heart failure, dyspnoea and orthopnoea are associated with a substantially greater amount of work than normal because the physical properties of the lungs are changed. Christie (1953) has reported in summary the interesting finding that the rate and depth of breathing are adjusted at rest or during exercise, so that the subject performs the minimum amount of work in moving the lungs by automatic adjustment of the rate and depth of breathing.

THE SUBDIVISIONS OF THE LUNG CAPACITY

The most commonly measured subdivision of the lung capacity is the vital capacity which was first studied by Hutchinson after he invented the spirometer. It soon became known that the vital capacity of normal subjects varied widely and that in certain diseases the maximum amount of air which could be drawn into the lungs and then expelled was small. We now know that after a maximal expiration a considerable volume of air remains in the lungs.

If a spirometric tracing of quiet breathing is made, it will be found that the depth of successive respirations is approximately the same (fig. 3); this is the *tidal volume*. The lower level of such a tracing, which represents the limit of normal expiration, is fairly constant and is referred to as the *resting respiratory (or end-expiratory) level*.

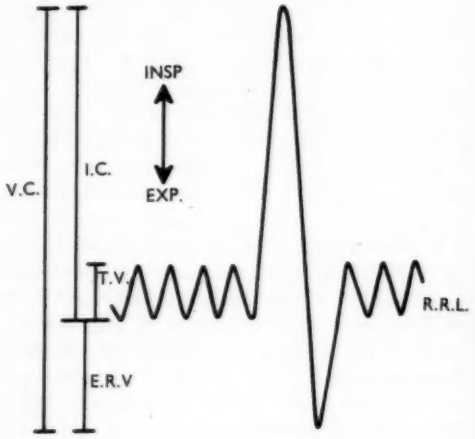

FIG. 3

Diagrammatic representation of a spirometer tracing showing vital capacity (V.C.), inspiratory capacity (I.C.), tidal volume (T.V.), expiratory reserve volume (E.R.V.), and resting respiratory level (R.R.L.)

The maximal volume of air which can be drawn into the lungs from the resting respiratory level is called the *inspiratory capacity*. The volume of air which can be expired after a normal expiration is called the *expiratory reserve volume*. The air which remains in the lungs after a maximal voluntary expiration is called the *residual capacity*, which is not shown in figure 3. The vital capacity is the sum of the inspiratory capacity and the expiratory reserve volume. The *total lung capacity* is the sum of the vital capacity and the residual capacity. The *functional residual capacity* is the

volume occupied by the expiratory reserve volume and the residual capacity.

The *vital capacity* is so easy to measure and is affected in so many pulmonary diseases that it has been extensively studied.

A measure of vital capacity can be used in two ways: either (1) to compare the subject's performance on one occasion with that on a subsequent occasion so that he acts as his own control, or (2) to relate the subject's performance to that of comparable normal individuals. Many attempts have been made to find a means of predicting the vital capacity, but none has been entirely successful.

Table I is taken from the work of Hurtado and Fray (1933) (with medical students) and of Aslett, Hart and McMichael (1939) (with coalminers) and shows the difficulty of prediction. They studied the degree of correlation between the vital capacity and certain easily determined body measurements.

The nearer the correlation coefficient is to unity the more closely can the vital capacity be predicted. Thus, the vital capacity had a high degree of correlation (+0.72) with radiological lung volume on inspiration (R.L.V. Insp.) in Hurtado's series and an appreciably lower correlation (+0.63) in McMichael's series. Differences are much more marked when the correlation of other pairs of variates are considered. For vital capacity and body surface area, for example, the figures were +0.28 and +0.67, which means that in the former series there was little correlation between these two variates, whereas in the latter correlation was moderately good.

The explanation of these divergent results is not far to seek. The two sets of measurements were made on different samples of men; both groups were clinically "normal," but their members were quite different in build, occupation and so on. A correlation coefficient between the vital capacity and another variate determined on a group of medical students cannot, therefore, be used to help in predicting the vital capacity of coalminers or any group of individuals other than medical students.

TABLE I

Correlation coefficients between the vital capacity and bodily measurements in adult males.

Characteristics correlated	Correlation coefficient
V.C. and R.L.V. Insp.	+0.72
*V.C. and R.L.V.	+0.63
V.C. and body-height	+0.55
*V.C. and standing-height	+0.70
*V.C. and stem-height	+0.78
V.C. and body-surface area	+0.28
*V.C. and body-surface area	+0.67

The correlations marked with an asterisk have been taken from the paper by Aslett, Hart and McMichael (1939); the others are from Hurtado and Fray (1933).

Inspiratory capacity and expiratory reserve volume.

The two components of the vital capacity, the inspiratory capacity and the expiratory reserve volume, are of interest because their relative magnitudes depend upon the position of the resting respiratory level.

When the tidal volume is increased voluntarily, or as a result of exercise, the increase is mostly at the expense of the inspiratory capacity. Hence a decrease in the inspiratory capacity results in dyspnoea at a lower level of exercise than in normal subjects.

The main interest in the expiratory reserve volume is that it forms, with the residual capacity, the functional residual capacity. This represents the volume of air in the lungs with which the inspired air mixes; it is upon the efficiency of this mixing process that replenishment of oxygen in the alveolar air depends. A large functional residual capacity, however, is not necessarily associated with an impairment of gas mixing and alveolar ventilation.

Functional residual capacity.

The method of determining the functional residual capacity has been described recently by Bates and Christie (1950). In principle it involves measuring the change in the concentration of an inert gas (such as helium) in a spirometer, when the functional residual air in the subject's lungs is allowed to mix completely with the gas in the spirometer. During the experiment the oxygen utilized is exactly replaced by oxygen admitted from a cylinder. A simple calculation, which takes into account the decrease in the concentration of helium in the apparatus and the known volume of the spirometer and its connections, gives the volume of the functional residual air. If the

volume of the expiratory reserve volume is subtracted from the functional residual capacity the volume of the residual air can be calculated.

A wide range of "normal" values is found for the subdivisions of the lung volume (table II). This means that a suspicion of abnormality is not aroused unless the volume of one or more of the subdivisions in a patient is grossly different from the corresponding mean value obtained from appropriate normal subjects used as controls. It is evident, therefore, that measurements of the subdivisions of the lung volume are, taken alone, unlikely to reveal small changes in lung pathology.

TABLE II

Relationship, in 50 normal young adult males, between the total lung capacity and its subdivisions. Total lung capacity = 100%

Fraction of lung capacity	Mean %	Standard deviation from the mean	Confidence limits* 67%	95%
Vital capacity	78.0	±4.3	73.7–82.3	69.4–86.6
Inspiratory capacity	61.9	±5.3	56.6–67.2	51.3–72.5
Expiratory reserve volume	16.2	±4.1	12.1–20.3	8.0–24.4
Residual capacity	22.0	±4.3	17.7–26.3	13.4–30.6

Columns 1, 2, and 3 have been taken from Hurtado and Boller (1933, their table 3).

*Confidence limits: 67%, means that 67% of normal young males would be found to have ratios within the limits shown in the table.

The following are references to some examples of groups of individuals who may be used as standards of "normality": Hurtado and Boller (1933) and Hurtado and Fray (1933) used medical students aged 18–30 years; Kaltreider, Fray and Hyde (1938) used males aged 18–30, males aged 38–63, and females aged 18–34 years. Whitfield, Waterhouse and Arnott (1950) studied subjects aged 10–70 years.

DYNAMIC MEASURES OF LUNG FUNCTION

Maximum breathing capacity.

It is usual when testing the function of any organ to subject it to some stress and measure the way in which it stands up to the strain. The exercise tolerance test and the urea concentration test are examples. So far as the lungs are concerned, the interest lies in the minute volume of ventilation which the subject can call upon and the efficiency of alveolar ventilation.

It is possible for normal individuals voluntarily to ventilate their lungs at the rate of 200 or more litres a minute for short periods of time. Patients with emphysema, on the other hand, may achieve a voluntary minute ventilation of no more than 20 litres. Hermannsen introduced the maximum breathing capacity test to provide a quantitative measure of differences in the power of voluntary ventilation.

To perform the test he used an ordinary spirometer from which the valves and carbon dioxide absorbing canister had been removed. Subjects were instructed to breathe into the apparatus as deeply as possible and as rapidly as possible for about 20 seconds, and a tracing of the movements of the spirometer bell was obtained. The volume of air, calculated in litres per minute, which the subject breathed out was referred to as his maximum breathing capacity (M.B.C.). Experience has shown that this test is a sensitive index of the mechanical function of the lungs.

D'Silva and Mendel (1950) reported that measurements of M.B.C. were not possible with their group of normal medical students using a spirometer of conventional pattern, because the inertia of the instrument was too great. Bernstein, D'Silva and Mendel (1952) published the design of a new spirometer which overcomes these difficulties and found that the M.B.C. of normal subjects is not reached at respiratory rates below 70 breaths a minute.

Various investigators have used specially designed valves, through which the subject's expired air during maximum breathing was passed into a Douglas bag for a known time. The volume

of gas in the bag was measured and the M.B.C. calculated. The success of the determination by this method largely depends upon the low resistance offered by the valves to the passage through them of rapidly moving gas. They are designed so that the maximal rate of gas flow (which may be well over 300 litres a minute) is hindered as little as possible. The greater the resistance offered by the valves, the poorer is the subject's performance in the test. The disadvantage of the method is that no graphic record of the subject's performance is obtained.

When the M.B.C. test was performed at a controlled respiratory rate, D'Silva and Kazantzis (1954) found that the tidal volume during maximum breathing in normal subjects was a fairly constant fraction of the subject's vital capacity and depended on the rate of breathing. Thus, in a series of 44 subjects breathing at 70 breaths a minute this fraction (called the swept fraction) was 52.2% ± S.D.7.22. At 30 breaths a minute it was 79.5% ± S.D.8.34. The value of relating a subject's performance in the M.B.C. test to his vital capacity is shown by comparing the performance of young normals with that of young patients with ankylosing spondylitis (D'Silva, Freeland, and Kazantzis, 1953). Although the M.B.C. of the two groups was widely different, their swept fractions were the same. This result means that in these patients the ease with which the lungs could be moved was the same as in normal subjects, though the magnitude of the excursion was limited by mechanical factors operating outside the lungs. The results obtained from patients were reproduced in normal subjects in whom the movements of the thoracic cage were restricted by a tightly laced waistcoat.

The prediction formula suggested by Wright and his colleagues (1949) which related a subject's performance in the test to his age and surface area, only approximately represented the performance of 14 normal medical students studied by Mendel (1949). He found that the M.B.C. of the men ranged from 140 to 320 (mean 203) litres a minute, whereas their predicted M.B.C. ranged from 154 to 171 (mean 165) litres a minute. Baldwin, Cournand and Richards (1948) have also published a prediction formula which gave figures for the same group ranging from 120 to 159 (mean 132) litres a minute. It is apparent that it is not possible to predict the M.B.C. of an individual with any useful degree of accuracy from his age and measurements of his physical proportions.

"Fast vital capacity" curves.

If a subject makes a maximal inspiration, and then a record is made of the rate at which this is breathed out when he exerts a maximum of effort, the curves, drawn from the original record and shown in figure 4, are obtained.

Curve A is from a normal subject, B is from a patient. Subject A breathed out his vital capacity volume in one-quarter of the time taken by B, as the curves show. The vertical lines *a* and *b* represent those portions of the respective vital capacities which the two subjects breathed out tidally at a respiratory rate of 30 breaths a minute. Subject A breathed out 73 per cent of his vital capacity compared with 32 per cent by B. As their respective vital capacities were 4.36 and 3.15 litres, it is obvious that A's performance in the test was much better than B's.

It might be, therefore, that accurate forecasts of M.B.C. would be possible from a study of "fast vital capacity" curves.

Gaensler (1951) has found that the percentage of the total vital capacity breathed out in the first second of a single maximal breath, exhaled as rapidly as possible, was, on the average, 83 per cent, and that the performances of normal subjects in the fast vital capacity test were closely correlated with their M.B.C.s. It is known, however, that the fast expiratory and the fast inspiratory curves are of quite different shape; Gaensler's method of prediction neglects the fast inspiratory curve. It is necessary, therefore, to extend Gaensler's observations to take into account both the fast inspiratory and the fast expiratory curves.

If a record of maximum breathing is made on a fast moving drum the inspiratory and expiratory portions of the tracing can be superimposed upon parts of the fast inspiratory and fast expiratory curves respectively. When the rate of maximum voluntary breathing is increased the tidal air is reduced, but the inspiratory and expiratory portions of the tracing can still be superimposed upon

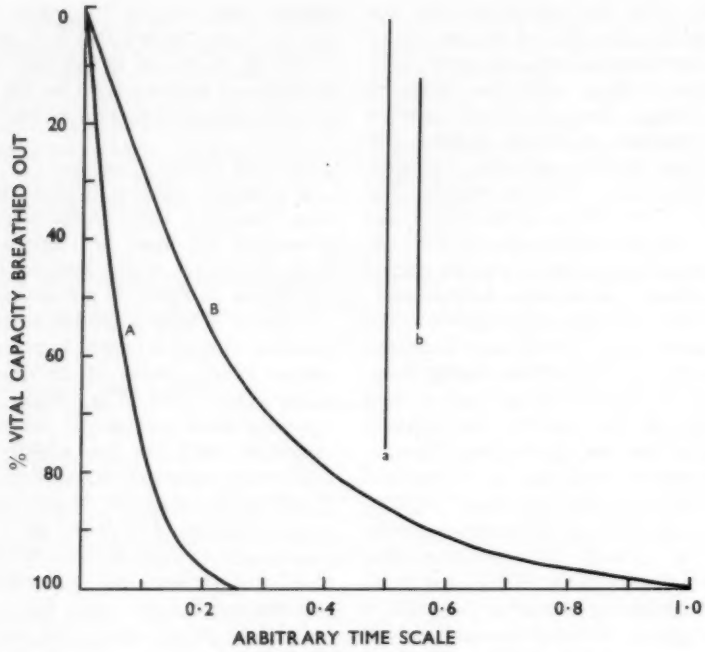

FIG. 4

Two fast expiratory curves (A and B) drawn on an arbitrary time scale; *a* and *b* are the tidal volumes of the two subjects breathing as deeply as possible at 30 r.p.m.

parts of the fast inspiratory and the fast expiratory curves. This means that it is possible, from records of the fast inspiratory and fast expiratory curves, to forecast the maximum voluntary ventilation of an *individual* at any respiratory rate. The method of doing this has been described by Bernstein and Kazantzis (1954). They found that the tidal air of a normal subject, expressed as a fraction of his vital capacity at any respiratory rate, could be forecast within a few per cent from measurements made on his fast inspiratory and fast expiratory curves. These observations have since been extended with similar results to a group of patients with various pulmonary diseases (Ogilvie, 1956).

CONCLUSION

The stage which has already been reached in research on the mechanical function of the lungs enables one to obtain moderately reliable quantitative data about the function of normal lungs *as a whole*. First steps in the extension of these investigations to a study of each lung have been possible since bronchospirometry was introduced. So far very little work has been done on the study of single lobes or of even smaller portions of the lung. Should this become possible, then physiological techniques may be expected to provide useful information about the magnitude of the disorders of mechanical function. At present the disorder must be considerable to be revealed by tests of lung function. The solution to the problem seems to lie in parallel studies of normal and diseased lungs. The purely "academic" studies on the former will lead to a more accurate interpretation of the results obtained on patients.

Needless to say, these various tests of pulmonary function can give no information about the

aetiology of lung disease. They may, however, be utilized in animals in which aetiological studies are being made.

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CORRESPONDENCE

THE ADMINISTRATION OF NEOSTIGMINE

Sir,—I welcome Dr. Lawson's report of cardiac arrest following the use of neostigmine (*Brit. J. Anaesth.*, 1956, **28**, 336). It once more draws our attention to the dangers of this drug. I believe that neostigmine is administered far too casually by many of us. Like so many faults in anaesthesia, we get away with it most of the time.

Dr. Boulton in his letter (*Brit. J. Anaesth.*, 1956, **28**, 449) mentions the value of the pulse rate as an indication of vagal tone present. Based on this observation I have adopted, for the last few years, a very rigid routine in the administration of this drug. It is as follows:

The patient's pre-operative pulse rate is recorded. If muscle tone is considered inadequate at the end of the operation, intravenous atropine sulphate is given, the dose depending upon the pulse rate at this time. If the pulse is below 80 per minute, 1/75–1/50 grain (0.5–1.3 mg) is given; if it is above this rate, 1/100 grain (0.65 mg) is given. The pulse rate is then taken at intervals until the maximum acceleration has been produced. Neostigmine (diluted to 2.5 mg in 5 ml) is injected slowly, in divided doses, until either adequate muscle tone has returned or the pulse rate has fallen to the pre-operative level. Should the muscle tone still be unsatisfactory when the pulse has slowed to this level, a further dose of atropine 1/100 grain (0.65 mg) is given before more neostigmine is administered.

This technique may sound tedious, but I believe it is safe, and, besides preventing the rare tragedy, it cuts down the incidence of salivation and abdominal colic.

I now teach this method to the students.

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THE PHARMACOLOGICAL ACTION OF ANAESTHETIC DRUGS ON THE CARDIOVASCULAR SYSTEM

BY

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THERE are few fields of anaesthetic work where more laboratory investigation with less immediate bearing on clinical practice has been carried out than that of the action of drugs on the heart and circulation. From the point of view of clinical anaesthesia, the pharmacological effects of anaesthetic agents on the heart and circulation must be considered from two entirely separate points of view. Firstly, there is the action of the drugs on vasomotor tone. Secondly, there are the effects on the cardiac musculature. These latter, because they are the more dramatic, have received attention which is far beyond their due. Fatalities due to the vasomotor effects of anaesthetics are, in fact, probably more common, and these will be considered first.

EFFECTS ON THE VASOMOTOR SYSTEM

All anaesthetic drugs depress to a greater or lesser extent the centres in the hypothalamus and medulla oblongata, which are concerned with the maintenance of vasomotor tone. In the normal individual the result is simply peripheral vasodilatation with a certain amount of flushing of the skin. Some degree of instability of the blood pressure will ensue, making the development of hypotension probable if the patient's head is raised high above the level of the rest of his body. In the patient who has already lost blood and whose blood pressure is being precariously maintained at a normal level by peripheral vasoconstriction, the administration of a general anaesthetic will always cause a fall in blood pressure. The amount of this fall varies with the agent used, but with some drugs the ensuing disturbances can lead rapidly to permanent irreversible circulatory failure. This point was first appreciated during the 1914–18 war, when it was realized that the administration of nitrous oxide and oxygen to wounded men was relatively safe, while the use of ether was dangerous and the administration of chloroform lethal (Cowell, 1919). The newer anaesthetic agents such as cyclopropane, trichlorethylene and vinyl ether are by no means free from the same unfortunate tendencies, while barbiturates are almost as equally lethal as chloroform. Indeed, rumour has it that at the time of Pearl Harbour lives were lost because of the indiscriminate administration of thiopentone by rule-of-thumb methods by technicians, as well as by the direct activities of the Japanese. There is, thus, clinical proof of the vasodilator effect of anaesthetic drugs, though these have been little studied in the laboratory.

ACTIONS ON THE HEART

The actions of anaesthetic drugs on the heart have been the subject of contention ever since that day in January 1848 when, three weeks after the discovery of chloroform, Hannah Greener died suddenly on the operating table in Newcastle. During the next seventy years a large number of investigations were carried out and none of them was frankly conclusive. It was, however, established that the heart could be affected in the laboratory in three different ways by anaesthetic agents. First, their administration could lead to a gradually increasing vagal tone, which would ultimately result in inhibition of the heart. Secondly, anaesthetic drugs could sensitize the myocardium to the effects of adrenaline, with the result that ventricular fibrillation with complete cardiac failure could be precipitated by the administration of this substance. Lastly, it was shown, mainly on the heart lung preparation of Starling or by simpler perfusion methods described by

Cushny (1936), that anaesthetic drugs in certain concentrations could cause arrest of the heart in diastole.

VAGAL EFFECTS ON THE HEART

Vagal effects on the heart occur to a certain extent with all anaesthetic agents. They were earliest demonstrated in a dramatic form by Embley in 1902, who was able to show that the administration of chloroform to a dog was followed by a progressive slowing of heart rate to the point of complete arrest, and that the administration of atropine or section of the vagi in the neck would protect his animals against this. Subsequently in his work on ethyl chloride Embley (1906) showed that the same phenomenon could occur when this agent was used. Much more recently, Haid (1954) and his co-workers in Iowa City have demonstrated, using the electrocardiograph, that vagal arrest of the hearts of experimental animals when ethyl chloride is given is an exceedingly common phenomenon. Vagal effects on the heart produced by cyclopropane have also been observed (Seevers et al., 1934), but these have been largely overshadowed and forgotten because of the interest in the tendency of this agent to produce ventricular fibrillation. Similar disorders occur with all agents in the laboratory, but the extent of the change is never so great as with ethyl chloride and chloroform.

Clinical Phenomena.

The clinical application of this work can be seen in the many papers on electrocardiography during anaesthesia (Lennox et al., 1922; Wachsmuth and Eismayer, 1928; Ward and Wright, 1929; Hill, 1932 a, b; Kurtz et al., 1936). In them there will be found records demonstrating the vagal effects of anaesthetic drugs; but because premedication with atropine has been almost universal since World War I, the florid and extreme type of cardiac inhibition noted by Haid and his colleagues (loc. cit.) in their animals is rare in man.

The increased vagal tone, however, associated with the induction of anaesthesia gives rise to simple bradycardia due to depression of the rhythmicity of the sino-auricular node by the vagus. The vagus also controls the conduction of impulses from the auricle to the ventricle. Heart block with an increase of the P–R interval on the electrocardiogram may therefore appear. This may progress to complete dissociation of the action of auricle and ventricle (fig. 1). In those more sensitive to agents acting on the vagal centres or where drugs with a special tendency to this action are given, the rhythmicity of the sino-auricular node may be entirely suppressed. In such circumstances the impulse to contract comes instead from the atrioventricular node, and, because its rhythmicity is so much less, the heart rate is correspondingly slowed (fig. 2). Even this structure may presently cease to provide enough impulses to discharge the rhythmicity of the ventricular muscle, and then idioventricular rhythm of very slow rate may appear. None of these disturbances is of serious significance in the adequately atropinized patient. If, however, the premedication has been mistimed or if the patient is submitted to a stimulus such as a major insult to the tracheobronchial tree, as when an endotracheal tube or a bronchoscope is passed, the process may go farther and a very marked slowing of the heart or even its complete arrest may occur. Even sudden inflation of the lungs in the unatropinized

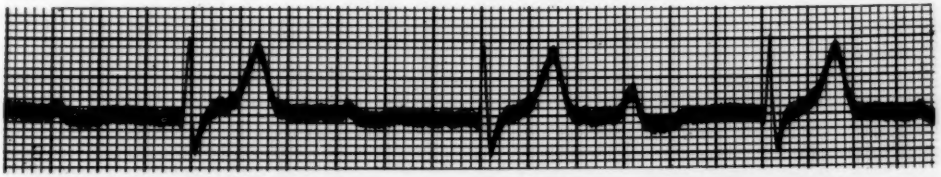

FIG. 1

A.V. dissociation under trichlorethylene anaesthesia. (By courtesy of Dr. J. Ives.)

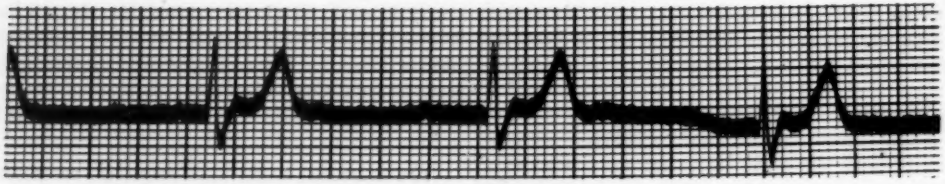

FIG. 2
A.V. nodal rhythm under trichlorethylene anaesthesia. (By courtesy of Dr. J. Ives.)

patient with an irritant vapour can have this effect —this is the pulmocardiac reflex of Johnstone (1951). Such disturbances are rare during anaesthesia. Waters (1951), however, in a study of patients to whom chloroform was given (carried out on the occasion of the centenary of the discovery of this agent) found that in three of the hundred patients he investigated, cardiac arrest occurred from vagal inhibition. Another agent which causes quite marked vagal hypertonia is trichlorethylene. This phenomenon appeared during the investigation of its action in human subjects carried out by Barnes and Ives (1944), but with this drug there is much less likelihood of progress to complete cardiac arrest, possibly because its action on respiration causes carbon dioxide accumulation which augments cardiac action.

EFFECTS ON VENTRICULAR MUSCLE

The second manner in which anaesthetics may affect the heart involves a direct action on the muscular wall of the ventricle. It is not clear whether this effect is truly exerted on the muscle or belongs more properly to the Purkinje fibres which are immediately under the endocardium. The point, however, is of academic importance. This type of disturbance was originally demonstrated in dramatic form by Levy (1911), who showed that the injection of a large dose of adrenaline into a cat lightly anaesthetized with chloroform was followed by a sudden exitus. A comparable effect was not obtained when the animal was deeply anaesthetized. At the time at which this work was done the electrocardiograph was not available, but shortly afterwards Levy (1913, 1914) extended his work and demonstrated beyond doubt that the process involved in the sudden cardiovascular collapse of his animals was the development of ventricular fibrillation. Subsequently, Beattie and his colleagues (1930) were able to show that sympathectomy, chordotomy or destruction of the centres in the hypothalamus which controlled circulation, would prevent the development of ventricular fibrillation when adrenaline was given to lightly anaesthetized cats. The risk of this complication under ether anaesthesia is negligible, and interest in it died down until cyclopropane came to be generally used in the 1930s. At first it seemed that irregularities due to this agent were related to the use of morphine for premedication (Robbins et al., 1939). More recently, however, it became apparent that anoxia and carbon dioxide accumulation from respiratory depression would more than account for the results of these early experiments. It was finally clearly shown by Waters and his colleagues working at Madison, Wisconsin (Meek et al., 1937), that serious cardiac irregularities could readily be produced by adrenaline in atropinized dogs under the influence of cyclopropane. A tremendous amount of work was done in this field in the years between 1939 and 1945, but the basic facts established by the Madison group remained largely unchallenged and still stand today. This group of workers demonstrated that the first effect of the administration of adrenaline to a dog under the influence of an anaesthetic agent which sensitized the myocardium was the appearance of ventricular extrasystoles. These quickly came almost completely to replace normal cardiac rhythm and a condition referred to in the American literature as "multifocal ventricular tachycardia" ensued. This was frequently followed within a matter of minutes by ventricular fibrillation (fig. 3). If fibrillation did not occur a spontaneous return of the cardiac rhythm

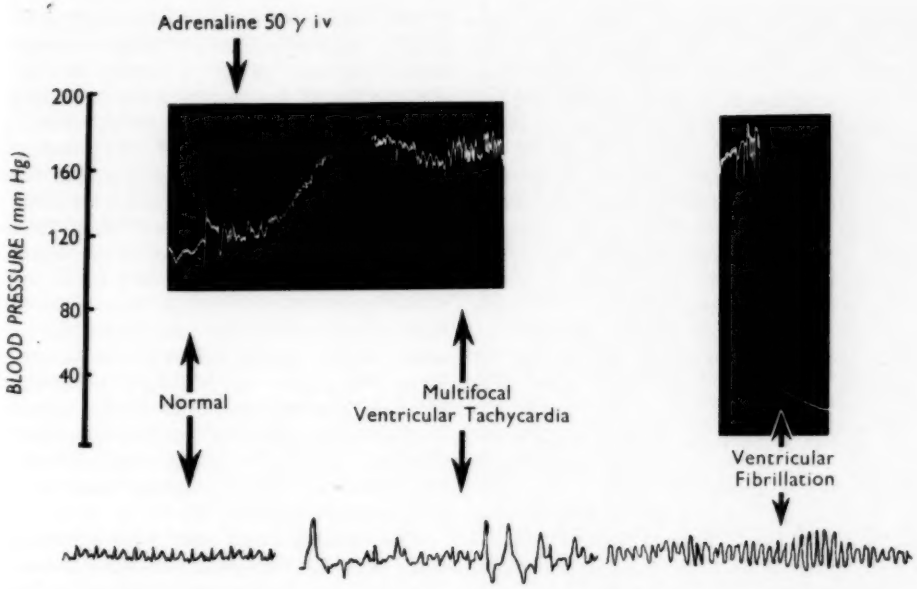

FIG. 3
Multifocal ventricular tachycardia and fibrillation in the cat under trichlorethylene after the administration of adrenaline. (Author: unpublished data.)

to normal took place and the animal was none the worse.

This work was originally undertaken for cyclopropane, but it has since been shown that sensitization of the myocardium to adrenaline with a liability, in the dog at least, to the development of ventricular fibrillation, is an accompaniment of the administration of ethyl chloride or trichlorethylene (Haid et al., 1954; Waters et al., 1943; Orth and Gillespie, 1945). It has also been established that procedures which stabilize the blood pressure at a lower level than normal, or at least prevent hypertension will largely forestall the development of these dangerous signs after the administration of adrenaline (Allen et al., 1940). Indeed, a simple mechanical contrivance which stabilized the blood pressure of an experimental animal was found in one laboratory to afford a very considerable protection against ventricular disturbances following the administration of adrenaline to animals under cyclopropane (Moe et al., 1947, 1948).

Clinical Phenomena

Clinically, ventricular fibrillation due to the anaesthetic is a much less common occurrence than is vagal arrest of the heart. None the less, minor indications of irritability of the ventricular muscle occur fairly regularly under anaesthesia with agents which show this phenomenon in experimental animals. Thus, Hill (1932 a, b), in one of the earliest studies of electrocardiographic changes under anaesthesia in the human subject, noted frequent extrasystoles in patients anaesthetized with chloroform. In the classic American study of the same subject Kurtz, Bennett and Shapiro (1936) noted ventricular extrasystoles, ventricular multifocal tachycardia and in one instance frank ventricular fibrillation, particularly in the patients to whom cyclopropane had been given. A large number of electrocardiographic studies of anaesthetized patients have been carried out since, but little has been added to our knowledge. Pulsus bigeminus has, however, been shown to be an early indication of ventricular

irritability (fig. 4). Johnstone (1950), too, has shown that carbon dioxide accumulation is as fruitful a cause of cardiac irregularities in man as it is in experimental animals. Another important contribution in this field was the paper of Barnes and Ives (1944), who noted ventricular extrasystoles with rather ominous frequency during trichlorethylene anaesthesia in the human subject (fig. 5). The anaesthetic agent isopropyl chloride, which for a time seemed as if it might be the agent of choice for tonsillectomy in young children, was rapidly abandoned after it had been shown that it gave rise to serious electrocardiographic disturbances indicating an increase in myocardial irritability (Elam and Newhouse, 1951).

The clinical recognition, without the electrocardiograph, of irritability of the myocardium produced by the anaesthetic agent is not easy. With some agents, for example trichlorethylene, extrasystoles appear as dropped beats or alternating with normal contractions, as pulsus bigeminus. In extreme instances the pulse becomes completely irregular. Possibly because of this tendency for the early development of warning signs, progress of the irregularities due to trichlorethylene to the point of frank ventricular fibrillation is rare in the human subject, though a recent publication suggests that it may not be quite so unusual as has been supposed (Edwards et al., 1956). On the other hand, in the case of cyclopropane, the sequence of events may be the development of a multifocal ventricular tachycardia with relatively little warning and the progress of the disturbance to fibrillation within a few minutes. Unfortunately, this complication seems to arise in healthy young adults and it is thought to be particularly liable to appear in the presence of hyperthyroidism where sudden and complete cardiac failure can occur within a few seconds. With chloroform the situation is exactly the same. Where ventricular fibrillation is going to occur it appears relatively suddenly. Indeed, in the old days it was all too often fatal and fully established before resuscitative measures could be taken.

The treatment of ventricular fibrillation is dealt with in other papers in this issue and in any case casts no light on the pharmacology of these drugs in the human subject.

When it was realized that in experimental

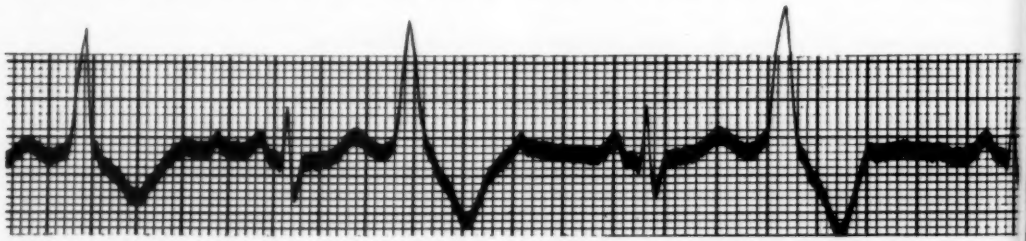

FIG. 4
Pulsus bigeminus under trichlorethylene anaesthesia. (By courtesy of Dr. J. Ives.)

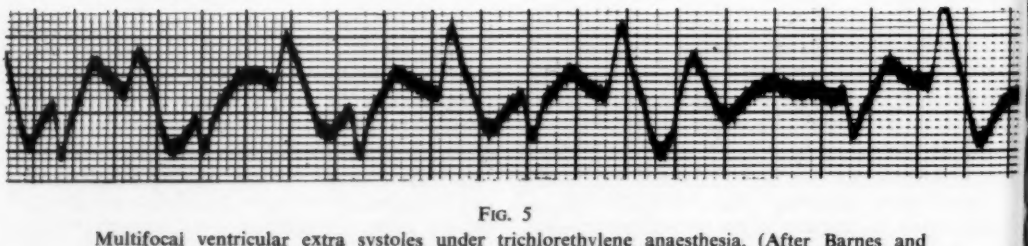

FIG. 5
Multifocal ventricular extra systoles under trichlorethylene anaesthesia. (After Barnes and Ives, *Proc. roy. Soc. Med.*, **37**, 528; by kind permission of the Royal Society of Medicine.)

animals under cyclopropane and similar agents, adrenaline could so easily precipitate ventricular fibrillation, the other sympathomimetic agents were investigated also. It was found that, without exception, they too were liable to precipitate ventricular fibrillation (Orth et al., 1939). Their use for the treatment of hypotension in patients under the influence of anaesthetic agents, which increase myocardial irritability, is therefore to be avoided. Recent work (Cummings and Hays, 1956) suggests that some sympathomimetic amines, notably phenylephrine, methoxamine (Vasoxyl) and naphazoline, are less dangerous than the other older agents in this respect. The pressor extract of the pituitary gland known as pituitrin or pitressin was also found to be liable to precipitate ventricular fibrillation in comparable circumstances (Morris et al., 1952a). Unfortunately, this fact for long failed to receive the publicity which was its due, and a large number of women were given this substance at the end of an instrumental or operative delivery under cyclopropane anaesthesia (Greene, 1942; Belinkoff, 1944). As would be expected, an appreciable and avoidable mortality ensued. Cocaine was also investigated and though it had itself no influence on the irritability of the myocardium, it greatly reduced the amount of adrenaline required to produce a fatal effect (Ruben and Morris, 1952).

When it became apparent that the irregularities of the heart which were liable to occur under cyclopropane anaesthesia were so readily produced by the administration of adrenaline it was only natural that adrenergic blocking drugs should be used to prevent them. In the experimental animal dibenamine (Nickerson et al., 1946), tolazoline (Priscol) (Nickerson and Smith, 1949), and rogitine (Morris et al., 1952b) have been used successfully either to reduce the incidence or completely to prevent the appearance of cardiac irregularities in animals under cyclopropane when adrenaline has been given. No doubt piperoxane would also be effective for this purpose. The therapeutic effect of these agents, however, in an animal whose heart had already become irregular is very much less satisfactory. Indeed, in a series of experiments carried out by the author (Hunter, 1950), dibenamine given to cats whose hearts had already become irregular under trichlorethylene anaesthesia more often precipitated ventricular fibrillation than relieved the irregularities.

Since it was believed that the cause of the difficulties in animals which developed irregularities under cyclopropane and similar anaesthetics was the increased irritability of the myocardium, it was reasonable that recourse should be made to agents known to have the power of preventing or abolishing other cardiac arrhythmias. Quinidine is the classic agent for this purpose, but similar properties are possessed by procaine (Burstein et al., 1940) and its amide. Given as prophylactic agents in the experimental animal, all these drugs have some action in preventing the development of increased cardiac irritability (Nickerson and Smith, 1949). On the other hand, their value in the treatment of the established condition is, both in the experimental animal and in man, somewhat in doubt (Stutzman et al., 1945). Further, both procaine and procaine amide can give rise to hypotension, which can be a troublesome complication, especially when operations have to be carried out on patients with cardiac disease. They can also produce quite serious disturbances of cardiac rhythm on their own (Long et al., 1949; Manchetti, 1952). Lastly, neither quinidine, procaine, nor procaine amide can safely be given in unlimited quantities. Sooner or later with increasing dosage they will themselves so poison the ventricular muscle that it will cease to respond to the impulse descending from the A.V. node, and complete cardiac standstill of toxic origin will occur.

The direct effect of anaesthetic drugs on the heart received a certain amount of attention in the early years of the present century and it was clearly shown by Cushny (1936) that chloroform in a concentration of 0.015 per cent in the fluid perfusing a rabbit's heart could cause arrest in diastole. The corresponding concentration of ether was 0.4 per cent. These figures were at the time taken to be indicative of the relatively greater toxic action of chloroform on the myocardium than that possessed by ether. However, a concentration of chloroform in the blood of 25 to 30 mg per 100 ml is necessary

for the production of anaesthesia in man. This is already in excess of the 0.015 per cent found by Cushny to arrest the heart. His observations are, therefore, of academic interest only and have not been made for any of the more recently described anaesthetic agents, though Krantz and his colleagues (1935) have shown that trichlorethylene is more toxic to the isolated heart of the experimental animal than is chloroform.

In summary, the effects of anaesthetic drugs on the circulatory system are threefold. First, there is the increased load on the heart which the vasodilator action of all anaesthetic drugs imposes. Secondly, all inhalation anaesthetic drugs possess the power of increasing the irritability of the vagal centres in the medulla oblongata, and in the inadequately atropinized animal or human subject they can cause cardiac arrest. Lastly, a certain number of anaesthetic drugs increase the irritability of the ventricular muscle. This shows itself most often in the development of ventricular extrasystoles, though in some cases the condition may progress to ventricular fibrillation. The precipitating factor in the development of this complication is usually adrenaline secreted in response to the trauma of the surgical procedure. Attempts have been made to prevent this complication by the administration of adrenaline blocking drugs or by giving agents which reduce the irritability of the cardiac muscle. Neither method is completely satisfactory in clinical practice and the only path to safety consists in sharply limiting the doses of agents known to increase cardiac irritability. By this means it should be possible to avoid those depths of anaesthesia at which ventricular fibrillation may occur.

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THE TREATMENT OF CARDIAC ARREST

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IN a proportion of cases of cardiac arrest following surgery and anaesthesia the occurrence is expected as a terminal event in a hopeless disease. Then, even though the patient may be in the operating theatre, treatment is neither reasonable nor desirable. However, it does not seem likely that most cases of cardiac standstill occurring in the theatre —or a number of those in the ward—can be placed in such a category, since the underlying pathology is not necessarily irreversible and survival can be expected if treatment is carried out efficiently and promptly.

A measure of the value of proper treatment can be found from recorded statistics. Stephenson and Hinton (1953), who analysed the records of 1,200 instances of cardiac arrest occurring in countries all over the world, found that 28 per cent survived the catastrophe as a result of treatment: furthermore, 17 per cent of the 132 cases which occurred outside the operating theatre were permanently resuscitated. The last figure is encouraging since it underlines the fact that success may follow immediate treatment, even though this is not necessarily carried out by the surgeon. Johnson and Kirby (1954) in the seven-year period 1946–1953 had a survival rate of just over 52 per cent in 19 cases occurring in the operating theatre, but they note that though resuscitation was attempted in some other cases in the wards, on each occasion at least five minutes had passed before it could be started, and the final results were all unsuccessful.

AETIOLOGY AND PREVENTION

In the treatment of any disease it is logical to look for a cause and, if possible, to remove this, while at the same time introducing more specific therapy. The causes of cardiac arrest can be broadly classified into two groups (*Lancet*, 1953). First, it may result suddenly from the effects of anaesthetic drugs or procedures, direct surgical stimulation of the heart or great vessels, or indirect stimulation from procedures which reflexly affect the heart. Secondly, it can result from an inadequate venous return to the heart, as in haemorrhage or shock. It must also be remembered that a diseased heart may cease to beat irrespective of surgery or anaesthesia, though perhaps currently with both, and that elective cardiac arrest may yet become a practical reality in clinical practice to facilitate heart surgery (Melrose et al., 1955).

In the literature much attention has been focused on the potential dangers of reflex cardiac arrest, particularly in inadequately atropinized patients, and on the ill-chosen and excessive use of anaesthetic drugs. But though these aspects of the problem are obviously of importance, oxygen lack, carbon dioxide excess and direct interference with the heart and great vessels are often found to be the underlying causes when case-histories are carefully analysed.

TREATMENT

Once cardiac arrest has been diagnosed, whether it be in standstill or ventricular fibrillation, and whatever the initial cause, the immediate treatment must be aimed at re-establishing a circulation with oxygenated blood within three and a half minutes. Time must not be wasted over the diagnosis, which in thoracic and abdominal surgery presents little difficulty since the heart can be visualized or palpated. In other cases it must be presumed when the pulse cannot be felt peripherally or in a major artery, the pupils are fully dilated and respiration, if previously spontaneous, ceases—usually with a typical gasping rhythm. However, cessation of respiration alone cannot be used as a diagnostic sign. The waxy colour of the patient, the absence of bleeding from the cut surface of an operation wound and the fact that

auscultation reveals no heart beat are all points of additional help.

The time interval between cardiac arrest and the death of nerve tissue in the central nervous system has been shown experimentally to be three and a half minutes (Weinberger et al., 1940), and there is ample clinical justification for accepting this period, though patients have survived intact for longer intervals. It can, however, only be completely acceptable on the basis that the patient is adequately oxygenated up to the moment of cardiac arrest. If hypoxia is present before this occurs, then severe neurological damage may result from even shorter periods of ischaemia than three and a half minutes (Cooley, 1950).

ARTIFICIAL RESPIRATION

Ideally, artificial respiration with 100 per cent oxygen and cardiac massage should be started at once and together, but, because in some cases it is necessary to operate to get at the heart, the value of immediate and adequate respiration must be emphasized. Not only does ventilation of the lungs supply oxygen and diminish the anaesthetic concentration of inhalational agents, but physiological evidence suggests that it can also provide for a few moments some sort of a circulation which is sufficient to reach the brain (Thompson and Rockey, 1947). Even a minimal cerebral circulation is of value in lengthening the critical period before anoxaemia produces residual damage. The effectiveness of such a pumping action may be increased if respiration can be carried out with both positive and negative pressure phases. Particular attention must be paid to the question of carbon dioxide elimination, since accumulation of this gas may be a prominent aetiological factor in the causation of ventricular fibrillation.

CARDIAC MASSAGE

That a qualitatively adequate circulation can be maintained by cardiac massage has been shown by many people (Wiggers, 1936; Dripps et al., 1948; Beck and Rand, 1949; Kay, 1951). Systolic pressures of 50 to 70 mm Hg have been recorded by intra-arterial catheter during cardiac massage in man (Deuchar and Venner, 1953) while the value of opening the pericardium to massage the heart directly is well known.

There is very little evidence that the heart may be extensively harmed by manual massage, though at postmortem localized petechial haemorrhages can occasionally be observed in the epicardium (Ruzicka and Nicholson, 1947; Ehrenhaft et al., 1951). If cardiac massage is carried out for a long period there may be a sufficient number of them to lead to persistent hypotension in the immediate recovery period, and even late cardiac failure (Milstein, 1956). On the other hand, cases of localized rupture of the ventricle—presumably in old infarcted areas—have been reported (Hurwitt and Seidenberg, 1953; Haight and Sloan, 1955).

There should be no controversy about the best approach to the heart for cardiac massage. This is undoubtedly the thoracic, though the presence of an abdominal wound may be accounted as an exception to this general rule. The advantages of a thoracic incision are a direct approach to the heart to facilitate massage, an ability to open the pericardium and to clamp the aorta just below the origin of the vessels supplying the brain, and the ease with which drugs can be accurately injected into the heart and electrical defibrillation carried out, should either be necessary.

The incision should be made at the level of the fourth or fifth left interspace lateral to the internal mammary artery, while the costal cartilages above and below the space should be cut to allow the ribs to spread more easily. Only a knife is needed. A rib spreader is helpful but not essential in the first place.

When an abdominal incision is already present then the heart can be massaged through the diaphragm or directly by incising the diaphragm and entering the pericardial cavity (Bailey, 1941). More recently a modified abdominal approach has been described which gives direct access to the heart without incising the diaphragm. The most inferior part of the anterior mediastinum is entered—after splitting the xiphoid process to get an opening into the peritoneum and retroperitoneal fascia attached to the back of this structure —by pushing the fingers up immediately behind the sternum (Neri and Stevenson, 1954).

Massage should be carried out with a gliding

motion from the apex to the base of the heart (Wolff, 1950). It can best be performed with two hands, alternately compressing and releasing the ventricles between them, and taking care not to use the fingertips—these can easily perforate an atrium—but rather the flat of the hand (McMillan, 1955). If the heart is very small then one hand should be used with the palmar surface of the fingers against the counterpressure of the thenar eminence, rather than the thumb (Milstein, 1956). A rate of about 60-80 a minute should be attempted, remembering that an operator can only be expected to perform this efficiently for about five minutes without a break. A relief must be ready to take over at intervals. With proper massage a systolic pressure of about 70 mm Hg can be expected, and this with an improvement in the colour of the patient, a diminution in the size of the pupils and the start of spontaneous respiration if the patient is not curarized, should be looked for as early evidence of effective therapy. It is at this point that stock can be taken of the situation and consideration given to the introduction of further measures of treatment. In the first place these depend upon the state of the heart—whether in fact it is at a standstill or in ventricular fibrillation.

CARDIAC STANDSTILL

It is most important to restore a normal heart beat as soon as possible since the rate of blood flow with cardiac massage only approximates to half that produced by a spontaneous beat (Johnson and Kirby, 1949). Thus after about 20 minutes of massage the aortic blood flow begins to decrease. The heart has, of course, a greater resistance to anoxia than the brain, but unless the coronary flow is quickly restored a healthy beat will only be produced with difficulty. It is therefore wrong to imagine that once artificial respiration and cardiac massage are started the emergency is over. Indeed such a supposition is far from being true since the chances of success progressively decrease as time passes, despite massage.

Success depends not only upon an adequate output from the heart but also upon the peripheral resistance, so that fluid replacement in cases of cardiac arrest secondary to haemorrhage is an essential part of the immediate treatment. Such treatment and the value of intra-aortic transfusion and vasoconstrictor agents in aiding the coronary and cerebral circulations in cases of primary cardiac arrest is discussed later in this paper.

At an early stage the blood that is expelled from the heart should be concentrated in the cerebral and coronary circulations (Wiggers, 1940). This can be done by occluding the aorta just below the origin of the left subclavian artery. When the abdominal approach to the heart is being used compression at the level of the coeliac axis is useful (Vetten et al., 1955). In either case when massage needs to be continued for periods in excess of 20 minutes it is a wise precaution to release the aortic clamp for a short period to reduce the chance of permanent damage to the liver or kidneys.

Drugs. If these immediate measures do not lead to the return of a spontaneous heart beat, then the use of drugs must be considered. As a generalization a flabby and dilated heart is more likely to need help than one which has tone but is not beating. In no circumstances, however, should any cardiac stimulant be used until massage has restored the coronary flow and oxygenated the heart muscle. The two drugs which are commonly used—calcium chloride and adrenaline—not only increase cardiac tone and prolong systole but they also increase excitability. Anoxia tends to potentiate cardiac excitability and increase the risk of ventricular fibrillation when these drugs are given in its presence.

Adrenaline is still the most popular choice, though it is probably more likely to produce fibrillation than calcium chloride. A dose of 0.2 ml of a 1:1000 solution (0.2 mg) should be given, and for convenience at injection this should be diluted in 2 ml of saline. Calcium chloride is best used in a 10 per cent solution and 2 ml injected (Kay and Blalock, 1951). Both drugs can be repeated in the doses described.

There is no general agreement about the best place for the injection, but when drugs are placed in the cavity of the left ventricle they will quickly be massaged into the coronary circulation and hence the muscle. At one time or another various other drugs have been favoured, such as isopropylnoradrenaline, barium chloride, and a mixture of adrenaline with procaine—the latter to counter

the risk of fibrillation (Lahey and Ruzicka, 1950). It has also been suggested that the drug injected is more valuable quantitatively as a means of raising the coronary artery blood pressure than as a stimulant (Alexander and Hewer, 1952). Practical experience suggests that the overall value of all these agents is doubtful and certainly secondary to the other modes of therapy described.

ARTIFICIAL PACEMAKER

The heart can be made to beat artificially by the application of an electric current to the region of the sino-auricular node. The current must be so small that it does not cause ventricular fibrillation, and of short duration, being repeated at a rate of about the normal heart beat or a little faster (Callaghan and Bigelow, 1951). Such an electric pacemaker can be made to work by external application, and has been used successfully in cases of cardiac standstill, though it is of little value in the presence of ventricular fibrillation (Zoll et al., 1955). Unless an external pacemaker is immediately successful the chest must be opened and cardiac massage started.

VENTRICULAR FIBRILLATION

Fibrillation may be present when cardiac arrest occurs or may be precipitated in a heart at standstill by the effects of drugs or even of massage. The best and most specific treatment is to pass an electric shock through the heart so that when the immediate contraction which this causes has passed, the whole muscle remains relaxed in asystole. Then, it is hoped, normal contractions will start either spontaneously or after a brief period of further massage. The experimental passage of a current through a mammalian heart was first tried by Prevost and Battelli in 1899, but the therapeutic possibilities of such a measure for fibrillation were demonstrated experimentally by Hooker and his colleagues (Hooker et al., 1933).

The voltage required to defibrillate the heart depends upon the electrical resistance of that organ and the duration of time for which it has stopped beating. Electrical resistance depends upon the size of the heart and the efficiency of the contact between it and the electrodes. These should be curved to fit the ventricles and covered with cloth damped in saline to prevent burning of the epicardium. Voltages ranging from 110 to 250 may be needed, an adult with a normal sized heart and arrest of brief duration usually requiring about 130-150 volts for 0.5 seconds. If the duration of each shock is too long then bubbles may form in the coronary arteries from excessive heat (Milstein, 1956). When a single shock is not effective in restoring a normal beat, then it should be repeated, and if this also fails a series of six at 1.5-second intervals tried (Wiggers, 1940).

It is possible to defibrillate the heart by the application of an electric shock to the outside of the chest wall (Zoll et al., 1956). Such an external defibrillator may perhaps have some application for emergency treatment of ventricular fibrillation in the ward following drug therapy or myocardial infarction. It would, however, have to be carried out immediately to be both effective and independent of cardiac massage.

Drugs. The value of drugs in the presence of ventricular fibrillation is controversial, but they should never take precedence over electrical defibrillation unless an apparatus for this purpose is not available.

Procaine depresses cardiac contraction and decreases excitability, while prolonging the refractory period of the muscle. It causes hypotension and may delay the start of a spontaneous beat. The dose is 50 to 100 mg or 5 to 10 ml of a 1 per cent solution injected into the cavity of the heart. The local injection of procaine into the area of the superior atriocaval junction may stop ventricular fibrillation and is used as a preventive measure in cardiac surgery, particularly under hypothermic conditions (Radigan et al., 1955). Potassium also reduces cardiac contraction and prolongs diastole, but the depression produced by a dose that stops fibrillation may be so great that a spontaneous beat cannot be produced after it even with the aid of stimulant drugs (McMillan, 1955). The dose is 5 ml of a 4 per cent solution of potassium chloride and this may need to be repeated. Paradoxically, even adrenaline may be indicated in the presence of fibrillation when the heart is weak and soft, and the chances of recovery are slight, unless a firm muscle can be produced (Milstein, 1956). Another drug which has been found helpful, particularly when fibril-

lation occurs during hypothermia, is neostigmine. It has also recently been reported that rewarming from the cold state may be necessary before a spontaneous beat can be restored (Husfeld and Secher, 1956).

Surgery. If a patient with stenosis of the aortic valve develops ventricular fibrillation, it may be necessary to relieve the obstruction before the abnormal rhythm can be effectively treated (Milstein, 1956).

TRANSFUSION THERAPY

When cardiac arrest is secondary to an inadequate cardiac input, as in haemorrhage or shock, rapid fluid replacement must be combined with massage. In such a situation there is much to recommend the intra-arterial rather than the intravenous route, and the vessel most easily cannulized is the aorta. The heart itself can be transfused, but there are no particular advantages in using this method and the procedure hampers massage (Stephenson et al., 1953). The principal advantage of an intra-arterial transfusion is the immediate rise in the coronary artery pressure that it causes (Veal et al., 1952).

Whatever the circumstances, blood is the best fluid to administer provided the potential dangers of stored blood are appreciated. The too liberal use of citrated blood may be dangerous since citrate is a cardiac depressant (Bruneau and Graham, 1943; Cookson et al., 1954). However, large quantities of blood would have to be given very quickly for this to occur. A more likely danger arises from the use of blood which has been stored for some time, since the plasma potassium level will have increased as this ion leaves the red cells. After three to four weeks a concentration of potassium which is lethal to the heart may accumulate (Melrose and Wilson, 1953). This danger is particularly likely when stored blood is infused into the aorta near the coronary artery inflow.

An infusion of l-noradrenaline is an effective way of raising the systemic pressure quickly, though it cannot be anything but a temporary substitute for blood if haemorrhage is the cause of the collapse (Churchill-Davidson, 1951). This vasoconstrictor may also be of benefit in maintaining the peripheral resistance when the heart commences to beat normally after massage, but the resultant blood pressure is very low (McMillan et al., 1952). It should be given by intravenous infusion of a solution made up by adding 4 ml of 1:1000 noradrenaline ($\equiv$ 4 mg) to 1 litre of saline at a rate sufficient to maintain an optimum blood pressure. Careful and continuous observation is necessary, and the very real dangers of excessive vasoconstriction must be appreciated.

Other factors which help to maintain the venous return to the heart are the use of a Trendelenburg position and the application of tight bandages to each leg and thigh (Woodward, 1952; Fowler, 1952).

Transfusions and vasoconstrictors are important ancillaries in the treatment of primary cardiac arrest, where their value is principally to aid in the maintenance of adequate cerebral and coronary circulations. However, from experimental evidence it seems likely that their use causes a rise in the pulmonary venous and right auricular pressures, thereby suggesting a tendency to precipitate pulmonary oedema. Since this complication frequently occurs when a spontaneous beat cannot be quickly restored, they should be used with care in these circumstances (Vetten et al., 1955).

CARDIAC ARREST DUE TO AIR EMBOLISM

With the increase in the number of operations on the left side of the heart the risk of air entering the coronary artery circulation and causing arrest is very real. This does not, however, necessarily constitute an irreversible problem, since experimental work in dogs shows that if a high enough pressure is produced in the coronary arteries the air can be forced through to the venous side. To do this the aorta must be clamped and manual massage applied to the heart (Geoghegan and Lam, 1953). It may also be useful to aspirate any air which is in the left ventricle, and to raise the aortic pressure still further by means of an intra-aortic transfusion given above the level of the clamp.

Air may reach the right side of the heart and the pulmonary circulation after entering a peripheral vein. Cardiac arrest usually occurs rapidly, though it is dependent on the quantity of air and the state of the heart. If the patient is quickly placed on his left side and air aspirated from the

right ventricle, cardiac arrest may be averted. If it has occurred, immediate massage may restore a normal beat (Durant et al., 1947; Hamby and Terry, 1952).

COMMENT

If cardiac arrest is to be successfully treated then the occurrence must be anticipated to the extent that facilities are always available for its immediate and proper correction. A sterile cardiac resuscitation kit should be kept in every operating theatre and recovery ward. Moreover, there should be a plan of action so that no time is wasted by inessential manoeuvres. The ultimate result for the patient will depend upon the brief interval between cardiac arrest and the restoration of an effective circulation, and every anaesthetist should know what can and what should be done in that period.

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THE ASSESSMENT OF THE CARDIAC PATIENT FOR ANAESTHESIA

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THE ASSESSMENT OF THE OPERATIVE RISK IN CARDIAC DISEASE

PATIENTS with cardiac disorders present the anaesthetist with three main problems which are related to (1) the operation itself, (2) the ability of the patient to withstand operation or any of its complications, and (3) the selection of the anaesthetic agent and technique.

Surgical aspects. The operation may be selective, allowing choice of time, place and personnel. It may be urgent, allowing little time for the patient to gain the benefit of any treatment, or it may be an emergency, such as haemorrhage, in which case those on the spot must take rapid decisions, and give such supportive treatment as they can while the surgeon deals with the immediate necessity. A close rapport between anaesthetist and surgeon, and their joint understanding of the physiopathology of heart disease makes for greater safety. For example, today many surgeons do not find a completely darkened theatre essential for endoscopies, thus making easier the detection of cyanosis. Similarly, fewer insist on high bridges and steep Trendelenburg positions which embarrass respiration. While smooth anaesthesia and satisfactory surgery may put little strain on a patient, difficulties may mean that complications are less easily withstood, and many patients who have survived operation have died in the postoperative period. Such conditions as ileus can be a great additional strain on a patient with heart disease. The abdominal distension impedes venous return by hindering diaphragmatic movement and by pressing on the great veins. Replacement of the fluid and electrolyte losses requires great judgment in order to avoid increasing circulatory difficulties. Potassium deficiency (fig. 1) predisposes the heart to ectopic rhythms (Bellet, 1955) and may precipitate digitalis intoxication (Lown et al., 1953). On the other hand, potassium excess (fig. 2) is dangerous, even to the healthy heart. Thrombophlebitis is an increased risk in patients with circulatory failure, and often exists unsuspected in bedridden patients. The pulmonary infarction which may result often leads to grossly impaired lung function, especially in mitral stenosis, and this risk should be remembered not only in the postoperative but also in the pre-operative period.

The patient and his condition. These must be assessed in relation to the proposed operation. The cardiovascular state may be such that even an urgent condition such as an operable neoplasm must remain untreated, but this is unusual, as, given time for treatment of such conditions as congestive heart failure or for the healing of a recent cardiac infarction, surgery can often be carried through with little increased risk. The control of cardiac rhythm and rate, the correction of sodium and water retention, the treatment of anaemia, the prevention of pulmonary infections, and weight reduction in obesity can so change the picture as to allow the completion of even radical surgery.

In operations on the heart, although there may be trauma, the abnormality is usually corrected,

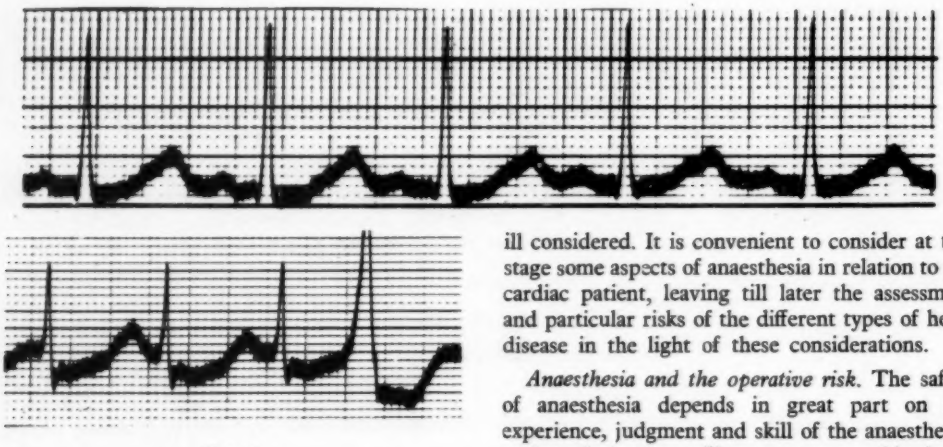

FIG. 1

Upper strip. Lead V5 of cardiogram of patient with hypokalaemia. There is slight depression of the ST segment, and large U waves which obscure the T waves.

Lower strip. Lead V5 from a similar patient with more severe hypokalaemia. There is marked ST segment depression, flattening of the T waves, and large U waves. There is also a nodal rhythm with retrograde P waves. One ventricular ectopic contraction can be seen.

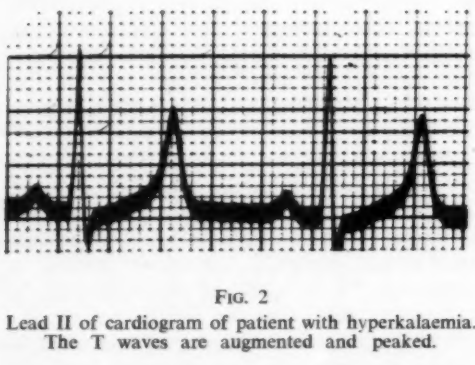

FIG. 2

Lead II of cardiogram of patient with hyperkalaemia. The T waves are augmented and peaked.

thus improving cardiac function. These patients usually come to surgery well prepared psychologically, anxious for any treatment and knowing only too well how little they have to lose. Such stoicism contrasts with the anxiety of patients undergoing minor procedures on other organs, who have been alarmed by warnings of the dangers of anaesthesia, which are usually unjustified and almost always ill considered. It is convenient to consider at this stage some aspects of anaesthesia in relation to the cardiac patient, leaving till later the assessment and particular risks of the different types of heart disease in the light of these considerations.

Anaesthesia and the operative risk. The safety of anaesthesia depends in great part on the experience, judgment and skill of the anaesthetist and to a lesser, but still great, extent on his choice of agents and method. A quiet confident manner together with a careful examination and a few comforting words of explanation will reduce the need for heavy sedation and may make possible the use of analgesic techniques, either local or general, which may carry a lower risk. Should heavy sedation be needed to reduce excessive oxygen demands or to prevent tachycardia, the gains must be balanced against the risks of depression of respiration and pharyngeal reflexes. A technique safe in a university department might require modification in different circumstances. When discussing the risks inherent in the use of various techniques and agents, we are well aware of excellent results achieved over many years employing methods which some would criticize on theoretical grounds. Usually the best work is done with the methods with which we are most familiar.

Hypoxia is the greatest danger to which the cardiac patient is exposed during surgical operations. It is often associated with other pathological conditions, such as heart failure or hypotension, either as cause or effect. Hypoxia must therefore be considered in relation to such states rather than as an isolated condition.

The heart removes nearly all the oxygen from the coronary blood as it passes through the heart muscle. The coronary sinus blood contains 4–6 volumes of oxygen per cent as compared with the 14 volumes per cent of oxygen in mixed

venous blood. In this it resembles other working muscles. It is, however, unable to incur an oxygen debt. For these reasons the heart is especially sensitive to hypoxia. When the arterial oxygen saturation begins to fall the healthy coronary arteries dilate in an attempt to maintain the oxygen supply to the myocardium; if the arterial saturation falls below 20 per cent the coronary flow is increased fivefold (Best and Taylor, 1945). The main factor regulating the coronary flow is the pressure in the aorta. If hypotension occurs in the presence of narrowed rigid coronary arteries the coronary flow diminishes. If hypoxia is added to hypotension even with normally dilating coronary arteries coronary insufficiency is probable. Tachycardia resulting from the effects of hypoxia tends to increase the cardiac output (Wiggers, 1949) and thus to compensate for the reduced oxygen tension. Excessive tachycardia shortens diastole, thus reducing the time available for ventricular filling and may actually diminish stroke output.

Ordinarily hypoxia is associated with carbon dioxide retention which in moderate excess causes tachycardia; gross carbon dioxide excess, however, impairs the conduction in the bundle of His, producing heart block and slow ventricular rate. Furthermore, carbon dioxide retention increases cardiac irritability and, especially in the presence of cyclopropane or chloroform, cardiac irregularity may be so gross as to impair the circulation. This effect is more marked when hypoxia co-exists. The hypertension associated with high carbon dioxide levels in the blood is the result of reflex vasoconstriction from the vasomotor centre. This centrally produced vasoconstriction is not obvious peripherally since the skin arterioles are relaxed by a direct effect. Should the carbon dioxide level in the blood fall after a period of increased tension, this vasoconstriction is replaced by vasodilatation with a resulting fall in blood pressure. This is accepted as the cause of "cyclopropane shock". In addition, the sudden fall of carbon dioxide, when the respiratory centre has been accustomed to a high level, leads to apnoea resulting in anoxia. This may prove fatal to the cardiac patient before the respiratory stimulus from the great vessels can restart the breathing. Hypercapnia is a much greater risk than acapnia which is unlikely to be seen in ordinary anaesthesia. It may, however, follow excessive and prolonged ventilation with high gas flows, efficient carbon dioxide absorption and muscle relaxants. This possibility should be kept in mind when artificial ventilators are employed for any length of time.

The association of anoxia and carbon dioxide retention is discussed in connection with pulmonary heart disease.

Hypoxia may also result from anaemia. The danger of circulatory overloading is well recognized, especially in heart conditions associated with left ventricular failure, mitral stenosis or pulmonary heart failure. Any transfusion to remedy the anaemia must be given slowly, and the use of packed red blood cells is advisable. The use of iron, perhaps given intramuscularly, may sometimes make transfusion unnecessary. Chronic reduction in blood volume occurs in many elderly patients, more especially in cases of malnutrition, sepsis or malignant disease. Hypovolaemia should be suspected in these states and the volume of transfusion increased to avoid severe hypotension.

In order to reduce the oxygen consumption of the tissues, hypothermia may be used, but it carries a greater liability to ventricular fibrillation with increasing age and in the presence of heart disease. This risk may have to be accepted in special cases to allow for the interruption of blood flow without causing irreparable anoxic damage. It has, however, a very small place in the anaesthesia of those with heart disease and then only in special centres accustomed to this technique.

On the other hand, the avoidance of hyperthermia, or even permitting a few degrees of cooling, is of considerable benefit. Every degree the temperature rises above normal requires an increase of 7 per cent in oxygen consumption. This is associated with peripheral vasodilatation and necessitates an increased cardiac output if the systemic pressure is to be maintained.

The blood pressure is maintained by the cardiac output and the total peripheral resistance. The total peripheral resistance depends on the state of constriction or dilatation of the arterioles. If these are dilated the blood pressure will fall

unless the output of the heart is increased. This increased output may be impossible in heart disease. As a fall in blood pressure below normal reduces the coronary filling pressure and hence the oxygen supply to the heart muscle, a further fall in cardiac output may occur—and a vicious circle is set up. This may be offset by the fact that both the work done by the heart and the pulmonary blood volume may be reduced. The two latter effects may be achieved by induced hypotension in cases of pulmonary oedema due to left ventricular failure. In general, however, most anaesthetists would avoid deliberately producing hypotension in cases of heart disease. The practice of increasing the bag pressure during hypotensive techniques in order to impede the venous return and hence to enhance the fall in blood pressure is open to criticism. When venous thrombosis, atherosclerosis, polycythaemia, or impaired liver or kidney function co-exist with heart disease, hypotension is especially to be avoided.

There is not yet agreement as to the circulatory effects of the generally accepted anaesthetic sequences. It would seem that with ether there is a release of adrenalin which causes tachycardia and a rise in blood pressure in the early stages, with a more normal level of blood pressure in the middle planes. There is some peripheral vasodilatation; the myocardium is but little affected until high blood concentrations are reached. It is true that cardiographic changes such as a wandering pacemaker and auricular ectopic beats may occur in light anaesthesia. These are probably due to vagal inhibition and are without ominous significance. Cyclopropane, like ether, causes some early reflex vagal inhibition but, unlike ether, it is also associated with more ominous irregularity of ventricular origin—ventricular ectopic beats, ventricular tachycardia and ventricular fibrillation. In contrast to ether, ventricular arrhythmia induced by cyclopropane increases as anaesthesia deepens and is associated with anoxia and CO_2 retention. They are thus increased by morphine which depresses respiration and are reduced by efficient ventilation.

Intravenous thiobarbiturates may produce a sudden fall in blood pressure. The pharmacologists consider that this is due to vasomotor depression but the authors feel that the possibility of an additional direct action on the myocardium cannot be excluded. Even ventilation with oxygen may not protect a severely ill patient from the direct circulatory effects of moderate doses of thiopentone (Edwards et al., 1956). The familiar sequence of thiopentone, nitrous oxide and oxygen is said to be accompanied by a rise in the peripheral resistance and a fall in the cardiac output (Elder et al., 1955). On the other hand, thiopentone in dogs has resulted in a fall in peripheral resistance and an increase in cardiac output (Greisheimer et al., 1956). Barbiturates do not appreciably increase the myocardial sensitivity to adrenaline.

Chlorpromazine has a strong anti-adrenaline action, and there is some evidence that it decreases the risk of ventricular arrhythmias. Peripheral vasodilatation, hypotension and tachycardia are, however, marked features of this drug's action.

The actions of various anaesthetic agents on the heart have received more attention than their effects on the peripheral circulation. These latter are of great importance. Animal work suggests that cyclopropane has the least effect on the mechanism regulating the peripheral vascular bed and that ether has a much more deleterious action; thiopentone has an intermediate effect. Even a healthy heart will fail if peripheral circulatory failure occurs (Hershey and Zweifach, 1950).

While there are difficulties in assessing the haemodynamics of anaesthetic agents in experimental animals and in healthy men, there is, for obvious reasons, very little precise information from patients with cardiac disease. Clinical impressions suggest, in general, a fall in peripheral resistance, tachycardia and a further fall in blood pressure; these imply an impairment of ventricular action. Many anaesthetists now feel, therefore, that all agents should be employed in minimal concentration. Fortunately muscle relaxants, which appear to have no toxic cardiac effects, now make this possible. Both induction and intubation can be made much easier by the proper use of relaxants, thus avoiding anoxia and carbon dioxide retention and hypertension. The incidence of arrhythmias during intubation and other intratracheal and laryngeal manoeuvres is increased by anoxia and is less often seen when thiobarbiturate, nitrous oxide and oxygen sequences are used in preference to ether or cyclopropane. Incidentally

it has yet to be shown that topical anaesthesia to larynx and trachea reduces the incidence of reflex disturbance during instrumentation. The importance of arrhythmias at this stage can easily be overemphasized even in cardiac disease. The same is not true for anoxia; experimental and clinical evidence combine to underline its danger. The use of an oximeter enables the oxygen saturation in the capillary blood to be watched during anaesthesia, and in particular it is instructive to observe the fall in oxygen tension during a clumsy intubation or an unduly prolonged tracheal toilet. Fink (1954) has discussed "diffusion anoxia" which is a risk on these occasions and at the end of operation. Then the nitrous oxide in the blood diffuses into the respiratory tract reducing the atmospheric oxygen tension. The remedy is ventilation with oxygen.

The use of relaxants implies maintenance of efficient ventilation. It is generally accepted that the tracheal pressures should follow the ideal curve described by Cournand et al. (1948). When using positive pressure ventilation the venous pressures are raised to maintain the flow to the right heart. This compensation may not be possible in conditions of reduced blood volume or "peripheral pooling". In these cases a negative phase interposed in the respiratory cycle is especially valuable (Hubay et al., 1954). These phenomena are most marked when the thorax is closed, but even with the open chest prolonged positive pressures are harmful and may produce hypotension (Papper and Reeves, 1947). Particular care in this respect should be observed when there is a right-to-left shunt in the heart which may be increased by positive pressure causing deterioration in the patient's condition.

After using relaxants it is, of course, important to be certain that full respiratory power has returned before allowing these handicapped patients to leave the theatre. Both atropine and neostigmine may have harmful effects on the heart. Atropine stimulates before it paralyses the vagus, and neostigmine, a potent anticholinesterase, produces strong vagal effects on the heart, resulting in bradycardia and varying degrees of heart block, up to and including arrest. These vagal effects are likely to be more dangerous in cardiac disease, especially in the presence of jaundice, cyclopropane or chloroform anaesthesia. While, therefore, it is better to avoid the use of neostigmine in these cases, there is absolutely no excuse for leaving such a patient underventilated. If neostigmine is needed for a cardiac patient it should be given, but the usual precautions are more than ever essential. The minimal dose of atropine for an adult is 1 mg (1/64 grain). This should be given slowly intravenously. At least 5 minutes should elapse before the neostigmine is given. If no tachycardia results, the atropine should be repeated before giving neostigmine. The neostigmine should be given in doses of about 0.5 mg, repeated at intervals until muscle power is adequate. During this period the patient must receive adequate oxygenation and the pulse rate must be carefully observed.

THE ASSESSMENT OF PATIENTS WITH SPECIAL REFERENCE TO DIFFERENT TYPES OF CARDIAC DISEASE

The assessment of patients with cardiac disease will now be considered in relation to specific types of cardiac disorder, bearing in mind that the risk of anaesthesia often depends as much upon the experience and skill of the anaesthetist and the pre-operative degree of cardiac efficiency as upon the type of heart disease.

COMMON CONDITIONS

Ischaemic heart disease.

The anaesthetic risk depends largely upon the severity of the ischaemia, being slight in patients with a small healed infarction (Etsten and Proger, 1955) and considerable in those with severe angina, marked cardiac enlargement, recent infarction or congestive heart failure. Two important factors must be considered. First the dangers of hypoxia and hypotension which predispose to further cardiac infarction, and second the tendency of the ischaemic myocardium to develop serious arrhythmias (notably ventricular tachycardia and ventricular fibrillation) which are likely to be induced by hypoxia. Special care should be taken to enquire for a history of recent cardiac pain or an increase in angina which may suggest a new infarction, and it should be remembered that increasing dyspnoea on exertion, paroxysmal dyspnoea, faintness, vomiting, or "indigestion"

may be the equivalents of the classical persistent pain of coronary occlusion (Parkinson and Bedford, 1928; Roseman, 1954; Papp, 1952). In examining the patient, cardiac irregularities due to ectopic contractions should be particularly looked for, and signs of impending or actual congestive cardiac failure in the form of gallop rhythm, raised jugular venous pressure, râles at the bases of the lungs, hepatic enlargement, and peripheral oedema, diligently sought. Care should be taken to examine the sacral region, as oedema commonly accumulates there initially in patients who have been in bed for more than a day or two. Many patients with ischaemic heart disease present no abnormal physical signs, the diagnosis being based upon the history and the electrocardiogram. Whenever possible, an electrocardiogram should be taken before anaesthesia, using at least three limb leads, and four praecordial leads, to detect evidence of recent infarction (fig. 3) or ischaemia (fig. 4). The occurrence of frequent ventricular ectopic beats (fig. 5) may denote the imminence of ventricular tachycardia and is therefore of sinister import and an indication for the prophylactic use of quinidine or procaine amide. Carbon dioxide accumulation should not be allowed to occur and anaesthetics which predispose to arrhythmias should be avoided if possible. If the history suggests a recent ischaemic episode, or the examination reveals signs of cardiac insufficiency, the anaesthetic should, if possible, be postponed while suitable therapy is instituted. If the operation is an emergency, aminophyllin intravenously and rapid digitalization may be indicated if there are signs of pulmonary congestion. Rapid auricular fibrillation (fig. 6) is an urgent indication for digitalization, but it should be remembered that intravenous

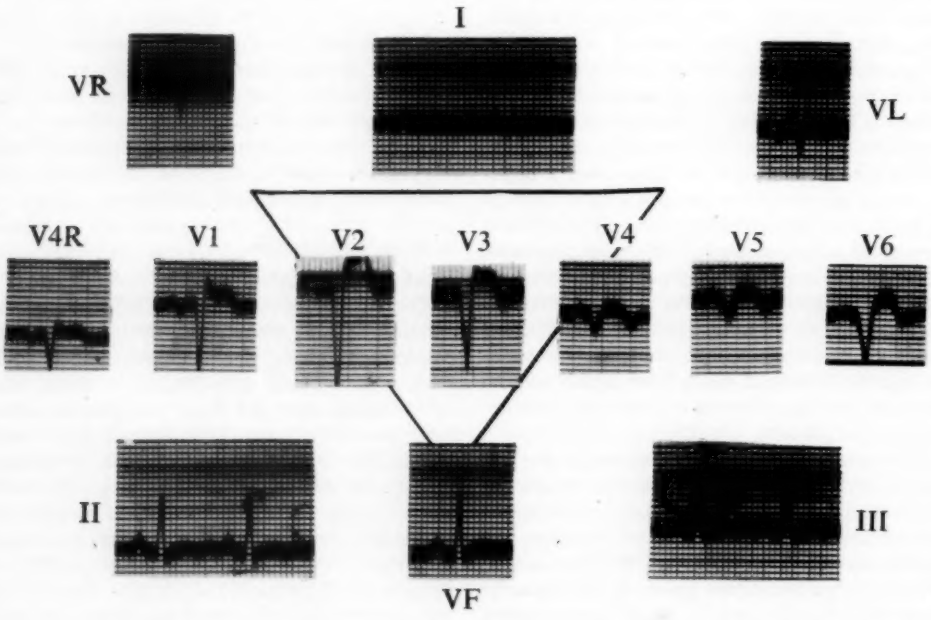

FIG. 3

Cardiogram showing recent extensive anterior cardiac infarction. There are wide deep Q waves in all chest leads, with ST segment elevation and T wave inversion. R waves are absent, suggesting through and through infarction. The duration of the QRS complex is slightly prolonged (0.12 sec) indicating peri-infarction block. (Note that standard leads show only minor changes.)

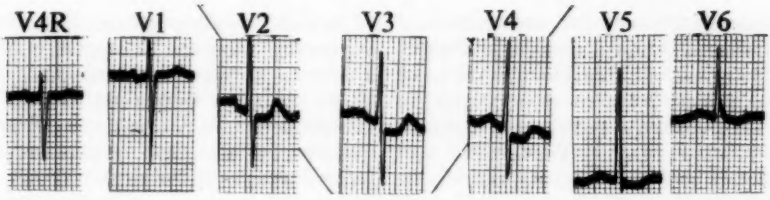

FIG. 4

Praecordial leads from the cardiogram of a patient with subendocardial ischaemia but no major infarction. There is ST segment depression in leads V2–6, and flattening of the T waves in leads V4–6.

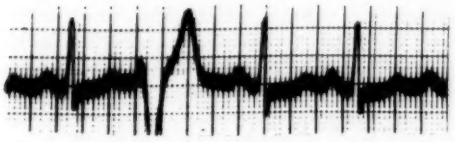

FIG. 5

Lead II of cardiogram showing a premature ventricular contraction, which falls at the end of the T wave of the preceding cycle. The QRS is widened and slurred, and the T wave augmented. There is no compensatory pause after the ectopic beat.

digoxin in full doses carries a risk of inducing ventricular fibrillation in patients with an irritable myocardium following a recent cardiac infarction.

Quiescent ischaemic heart disease with good cardiac function should not be allowed to stand in the way of important surgical proceedings, for Etsten and Proger (1955) found the operative mortality only 1 per cent higher in a group of patients with coronary artery disease than in a normal group.

Elderly patients without obvious heart disease may develop rapid auricular fibrillation during or shortly after anaesthesia, and also require digitalization.

Rheumatic heart disease.

Many patients with chronic rheumatic endocarditis have excellent cardiac function, and are often symptomless. In such circumstances no special precautions are necessary. Certain types of patients, however, require careful assessment, and special precautions before and during anaesthesia. Patients with severe mitral stenosis and pulmonary hypertension are subject to pulmonary oedema, sudden death due to acute right ventricular failure, and thrombo-embolism of the pulmonary arterial tree (Wood, 1954; Davies et al., 1954; Ball et al., 1956). Such patients have high left auricular and pulmonary venous pressures and frequently severe pulmonary arterial hypertension due to an increased pulmonary arteriolar resistance (Lewis et al., 1952; Wood, 1954). Attacks of pulmonary oedema may follow undue exertion, emotional stimuli, or fever. Excessive tachycardia is particularly dangerous, since these patients cannot increase their cardiac output, and the shortened ventricular diastolic filling time leads

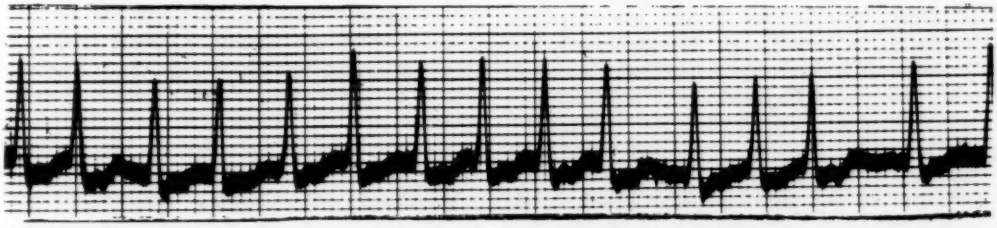

FIG. 6

Lead I of cardiogram showing rapid auricular fibrillation. The ventricular rate is completely irregular, and the normal P waves are replaced by irregular f waves. The QRS is normal.

to an increase in left auricular and pulmonary capillary pressure, which predisposes to pulmonary oedema (Lewis et al., 1952). In our experience pre-operative anxiety has been known to initiate an attack of pulmonary oedema in a patient with severe mitral stenosis. The patient with extreme stenosis may be recognized clinically by the apprehensive facies, cold extremities, peripheral cyanosis, and small pulse due to the low cardiac output. Examination of the jugular venous pulse often reveals a "flicking" pulsation just preceding the carotid pulse—the augmented "a" wave which results from the right auricular hypertension secondary to pulmonary hypertension. The latter can also be inferred from the accentuated pulmonary component of the second heart sound; this should not be confused with the opening "snap" of the mitral valve which is often well heard in the pulmonary area. The signs of mitral stenosis are too well known to need reiteration, but it is worth noting that when the cardiac output is very low the murmurs may be almost inaudible, which increases the difficulties of diagnosis. Great care should always be taken to minimize apprehension, to ensure adequate premedication, sedation, and smooth induction of anaesthesia, which may sometimes be profitably performed in bed. Hypoxia is especially to be avoided, and while care must be taken not to overload the patient with intravenous fluids, rigid dehydration should not be practised as it predisposes to salt deficiency and to peripheral venous, and pulmonary thrombosis in the post-operative period. Auricular fibrillation is extremely common in mitral stenosis, and must be supposed to indicate more advanced disease than does sinus rhythm. It is of interest that patients with extreme pulmonary hypertension do not always develop fibrillation until a late stage. The arrhythmia may be induced, however, by anaesthesia, and the onset of auricular fibrillation is an urgent indication for digitalis, which can be given intravenously in full doses, provided none has been given recently, without the risk of ventricular tachycardia which is present in patients with recent cardiac infarction. Well-controlled auricular fibrillation confers no added anaesthetic risk other than the possibility of embolism which can occur at any time.

Patients with severe rheumatic valvitis and myocarditis who are in congestive heart failure constitute greater anaesthetic risks, and every effort should be made to relieve the congestive failure before the anaesthetic is given. Such patients are intolerant of the over-enthusiastic administration of intravenous fluids, especially saline, and a persistently elevated jugular venous pressure and râles at the bases of the lungs are important warning signs. Patients with severe aortic stenosis are notoriously liable to sudden death (Lewes, 1951) and are vulnerable to hypotension and hypoxia, which may initiate ventricular fibrillation. A history of frequent syncopal or anginal attacks, increasing dyspnoea, and evidence of right ventricular insufficiency are ominous portents. Free aortic regurgitation may be of syphilitic origin and associated with aortitis and coronary ostial disease. In these circumstances the avoidance of hypotension and hypoxia is equally important.

Pulmonary heart disease.

Pulmonary heart disease may be acute or chronic. The former is typified by pulmonary embolism and the latter is most frequently caused by chronic bronchitis and emphysema. Such patients may have both respiratory and cardiac insufficiency, the former resulting in carbon dioxide retention and hypoxia, which leads to pulmonary hypertension, hypervolaemia and polycythaemia, and an increase in cardiac output (McMichael and Sharpey-Schafer, 1944; Harvey et al., 1951). Von Euler and Liljestrand (1946) showed in cats, and Motley et al. (1947) in man, that the pulmonary artery pressure rises with hypoxia, probably as a result of constriction of the pulmonary arterioles, although the rise in cardiac output also contributes. Progressive and recurrent hypoxia and distortion of the pulmonary vessels by emphysema or fibrosis results in constriction and obliteration of the pulmonary vascular bed and an increase in pulmonary vascular resistance. This causes persistent pulmonary hypertension, and ultimately right ventricular failure follows. Patients with chronic respiratory disorders are particularly liable to repeated attacks of bronchial infections which rapidly lead to anoxia, hypercapnia and right heart failure.

Mounsey et al. (1952) have shown that the pulmonary artery pressure rises during an attack of heart failure, and falls when the hypoxia has been relieved, the work of the right ventricle being reduced, thereby relieving the heart failure. In assessing the patient with chronic cardiorespiratory insufficiency special care should be taken to elicit a history of any recent increase in cough, dyspnoea, or wheezing. The production of purulent sputum and onset of fever also should suggest a recent acute pulmonary infection, and contra-indicate anaesthesia—unless urgently required—until treatment has been carried out. The appearance of the patient in the early stages of heart failure of pulmonary origin, when the cardiac output is relatively high (McMichael, 1948) is characteristic. Cyanosis is combined with a warm, moist skin, and a bounding, rapid pulse. The jugular venous pressure is raised, and signs of right ventricular hypertrophy and pulmonary hypertension are frequently found. There may be hepatic enlargement and peripheral oedema. Clubbing of the fingers is an important sign. In severe heart failure the respiration is often rapid and may be shallow and ineffective. Scott (1920) has shown that in emphysema the normal response of the respiratory centre to carbon dioxide is reduced. Hypercapnia then depresses the respiratory centre, the normal stimulus to respiration being provided by the low arterial oxygen saturation. When the disorder is severe the patient may be irrational, drowsy and irritable, and may have frequent muscular twitchings. These manifestations are the results of hypercapnia and hypoxia acting on the cerebrum (Westlake et al., 1955). It is in such patients that the uncontrolled administration of oxygen may produce respiratory failure, coma and death (Davies and Mackinnon, 1949). Patients in such advanced stages of respiratory insufficiency can scarcely be considered for anaesthesia, which may, however, be required for patients with milder forms of chronic chest disease.

Several points must be borne in mind in assessing the anaesthetic risk to patients with chronic cardiopulmonary disease. The importance of clearing up recent chest infections has already been mentioned, and appropriate chemotherapy, postural drainage, and antispasmodic therapy should be instituted before anaesthesia whenever possible. Patients with chronic asthmatic bronchitis may die suddenly if rendered hypoxic or if the respiratory centre is depressed. Morphine in such circumstances is absolutely contra-indicated, as it is in patients with severe thoracic deformities, such as kyphoscoliosis (Daley, 1945). Every effort should be made to keep the airway of such patients as free as possible from bronchial secretions during the operation, and bronchoscopy with suction is an important procedure at the end of the operation, provided that it does not itself exacerbate the hypoxia it was intended to reduce. Postoperative chest complications are greatly to be feared, and an energetic programme of breathing exercises, expectoration and chemotherapy should be arranged.

Thyroid heart disease.

Thyrotoxicosis commonly affects the heart in older patients, rapid auricular fibrillation being common, and not infrequently leading to congestive heart failure if uncontrolled. The classical signs of thyrotoxicosis—tremor, sweating, exophthalmos, loss of weight, and large diffuse goitre—may be largely absent in older patients who usually have a nodular goitre, which may be quite small. Unexplained auricular fibrillation, therefore, should always be suspected as thyrotoxic in origin, and a careful search made for symptoms and signs pointing to this diagnosis. Anaesthesia is poorly tolerated in patients with uncontrolled thyrotoxicosis, and it is noteworthy that the response of auricular fibrillation to digitalis is often poor until the thyrotoxicosis has been effectively controlled.

Hypertensive heart disease.

Hypertensive heart disease presents no special problems unless heart failure is imminent or present. Enquiry should be made into a history of paroxysmal or exertional dyspnoea. Left ventricular failure is not associated with peripheral oedema unless it is complicated by failure of the right ventricle. Therefore, attention should be paid to the presence of gallop rhythm (especially early diastolic gallop due to the addition of a third, or rapid filling sound; and to summation gallop which is due to both auricular and third heart sounds summating in mid-diastole), râles at

the bases of the lungs, and orthopnoea. The jugular venous pressure will usually be elevated, but not necessarily grossly so. Such signs are usually accompanied by a heaving apex beat outside the mid-clavicular line, which denotes left ventricular enlargement. The X-ray of the chest shows left ventricular enlargement, prominent pulmonary arteries, haziness of the lung fields, and often pleural effusions or shadows of pulmonary oedema.

The presence of these signs is a warning which should not be neglected, and treatment by salt reduction, mercurial diuretics, and digitalis is indicated. Often a dramatic and rapid reduction in the symptoms and signs of cardiac failure may be obtained by the injection of a potent hypotensive agent such as hexamethonium (Smirk, 1954).

LESS COMMON CONDITIONS

Congenital heart disease.

The commonest form of congenital heart disease associated with central cyanosis (due to admixture of venous with arterial blood within the heart) is the tetralogy of Fallot. This consists of a high ventricular septal defect, pulmonary stenosis, overriding aorta, and right ventricular hypertrophy. There is usually a history of cyanosis at or shortly after birth, dyspnoea and squatting on exertion. This last symptom is a valuable diagnostic pointer, since it is almost unknown in conditions other than those with pulmonary stenosis or atresia and a right-to-left shunt. On examination, the diffuse cyanosis and clubbing of the digits, the tapping right ventricular cardiac impulse, loud systolic murmur over the pulmonary artery and soft pulmonary valve closure sound, proclaim the diagnosis. Additional clinical pointers are the presence of associated skeletal deformities, deafness, hypertelorism, cataract, etc. Radiology of the chest reveals oligaemic lung fields with little cardiac enlargement. Sometimes difficulty may be experienced in distinguishing pulmonary heart disease with cyanosis and clubbing, from cyanotic congenital heart disease. However, the history of chronic respiratory disease, the overactive circulation, and signs of bronchitis, purulent sputum, and lack of notable cardiac murmurs will usually permit differentiation at the bedside.

Patients with the tetralogy of Fallot tolerate anaesthesia surprisingly well (with the exception of thiopentone other than in small doses), provided that plenty of oxygen is available. Morphine in small doses is not contra-indicated, but large doses of barbiturates are poorly tolerated. Controlled hypothermia is a valuable aid to anaesthesia.

Such patients have polycythaemia, and are prone to develop venous thrombosis, particularly in the brain. Care should be taken, therefore, to prevent further haemoconcentration and ensure adequate fluid intake after the operation. A smooth induction is important, as struggling and excitement is associated with an increase in the right-to-left shunt, and acute right ventricular failure may occur on effort (Howarth and Lowe, 1953).

Acyanotic conditions include arterial and ventricular septal defects, patent ductus arteriosus, and coarctation of the aorta. Small atrial septal defects may be symptomless, and difficult to diagnose clinically, since there may be little on examination of the heart other than a soft systolic murmur over the pulmonary artery, and a widely split second heart sound which does not alter on inspiration. The X-ray shows overfilled lungs and enlargement of the right auricle, right ventricle and pulmonary artery. The aorta is small. The electrocardiogram shows some form of right bundle branch block. There are often certain physical anomalies, the most important of which are a high arched palate, long limbs, hyperextensibility of the joints, arachnodaktyly and long patellar ligaments. These anomalies (which are sometimes associated with dislocation of the lens, and cataract) constitute Marfan's syndrome. This syndrome may be found with any other form of congenital heart disease, or anomalies of the aorta, such as medial necrosis, coarctation, and lesions of the aortic valve. Detection of the skeletal manifestations of Marfan's syndrome should arouse the suspicion of a cardiac or aortic defect, which should be diligently sought, the more so because sudden death is not uncommon.

Patent ductus arteriosus is readily recognized by the wide pulse pressure, and continuous murmur over the pulmonary artery. As with other lesions associated with a left-to-right shunt, pulmonary infections are not uncommon, and special pre- and postoperative prophylaxis may be required.

Most patients with coarctation of the aorta are only moderately hypertensive and constitute no additional anaesthetic risk unless severe associated anomalies, such as aortic stenosis and incompetence, are present in addition.

Heart block.

Patients with a labile atrioventricular block, and a history of Stokes-Adams attacks, constitute an anaesthetic hazard, although successful surgery and anaesthesia have been carried out in many such cases without disaster (Penton et al., 1956). All drugs which depress cardiac conduction must be rigorously avoided—especially digitalis, quinidine, and procaine amide. Sympathomimetic drugs, such as adrenaline, isopropyl noradrenaline, and vagal antagonists such as atropine should be at hand. The use of molar lactate has recently been advised (Bellet et al., 1955; Swash and Wallace, 1956). Stokes-Adams attacks commonly occur where partial atrioventricular block becomes complete but may also occur in patients with apparently established complete block. Occasionally attacks may be preceded or terminated by ventricular tachycardia, and they present a different therapeutic problem.

Enquiry into the occurrence of "fainting" attacks, or convulsions, followed by reactive flushing, should be made if heart block is suspected, and a cardiogram taken.

Constrictive pericarditis

This condition can usually be recognized clinically by the small heart, soft heart sounds, tachycardia, triple rhythm, and raised venous pressure. The heart is irritable, and auricular fibrillation is common, and may be induced during anaesthesia. Thiopentone is contra-indicated since peripheral vasodilatation and hypotension may prove disastrous.

In general the risks to which the patient with cardiac disease is exposed, depend on the nature of the proposed operation and its possible complications and on the general cardiovascular status of the patient.

The type of the anaesthetic, provided it is competently administered and conforms to basic principles, together with the specific nature of the cardiac disability is usually of lesser importance. Nothing overrides the truth that techniques and disease processes which impair the oxygen supply to the heart are always a threat to life.

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BOOK REVIEW

Anesthesia for Obstetrics. By Robert A. Hingson, Professor of Anaesthesia, Western Reserve University, and Louis M. Hellman, Professor of Obstetrics and Gynaecology, State University of New York. Published by J. B. Lippincott Company, Philadelphia. Pp. 344. Price £5.

Dr. Hingson and Dr. Hellman have approached this work with the intention of producing a text which accepts and defines the intimate relationship between anaesthesia and obstetrics, and since each is a recognized leader in his own subject, the reader can expect both an authoritative and well-knit whole. Nor will he be disappointed, as there can be very few books in which every aspect of obstetrics necessitating anaesthesia is so completely dealt with and yet in a way which shows thorough understanding of the problems of both subjects.

Throughout the work there is a degree of conformity with the ideas and practice which are familiar to doctors in this country, and an up-to-date and provocative approach to the challenge of modern drugs and methods. One fancies that the authors are well aware of the deficiencies associated with a too constant reliance on well tried but not always perfect remedies. If there appears to be some emphasis on the advantages of local analgesia, the reader might well interpret this as undue conservatism on his part rather than transatlantic bias. It is, in any event, well offset by the helpful and progressive attempts of the authors to illustrate the value of cyclopropane and trichlorethylene in obstetrics.

This book can be recommended for many things, yet its principal asset is the excellent understanding that so obviously exists between obstetrician and anaesthetist. There are some minor points of detail and some of practice which can be criticized, but none detracts from what is a useful and well produced volume.

W. D. Wylie

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ELECTROCARDIOGRAPHIC INTERPRETATION

BY

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THE electrocardiogram is a recording, made from the surface of the body, of the electrical currents produced in the heart during contraction. It must be realized that electrocardiography is still to a large extent an empirical subject but with increasing knowledge it is being built up on a more scientific basis. It is not necessary, in a short paper such as this, to discuss in detail the electrical changes which take place in the heart muscle since these are described fully in practically all textbooks of electrocardiography and cardiology. Wood (1956) has given an account which is easily followed and which will be of considerable value to those who wish to understand the fundamentals of what, at first sight, appears to be a rather complex subject.

The first leads to be introduced were the three standard or bipolar limb leads which record the difference in potential between two points, the electrodes being connected to the right arm, left arm and left leg as follows: Lead I, right arm and left arm. Lead II, right arm and left leg. Lead III, left arm and left leg. In a similar manner bipolar chest leads may be recorded, one of the electrodes being applied to the chest wall overlying the heart while the second is attached to the right arm, left arm or left leg. The chest or exploring electrode is given the letter C and the limb electrode R, L or F according to whether it is connected to the right arm, left arm or left leg. The chest electrode may be moved over the precordium, thus "exploring" the walls of the underlying right and left ventricles. The electrocardiographic pattern which one obtains will obviously vary according to the position of the exploring electrode in relation to the cardiac chambers, and it has, therefore, been necessary to select fixed points on the chest wall, and these were agreed upon by the Cardiac Society of Great Britain and Ireland and the American Heart Association. The six most common sites selected for routine recordings are as follows:

Position 1. The 4th intercostal space at the right border of the sternum.
Position 2. The 4th intercostal space at the left border of the sternum.
Position 3. Midway between positions 2 and 4.
Position 4. In the midclavicular line in the left 5th intercostal space.
Position 5. In the left anterior axillary line on the same horizontal level as 4.
Position 6. In the midaxillary line on the same horizontal level as 4.

Thus, if the chest electrode be placed at position 4 with the limb electrode on the right arm, the resulting lead is designated CR4. If, however, the limb electrode be placed on the left leg the lead is termed CF4. The CR leads tend to lay stress on the upright deflections while the CF leads stress the downward deflections. These leads may, therefore, in certain cases of myocardial infarction, be more valuable than the V leads described below.

As has already been stressed, these bipolar leads record the difference in potential between two points. Wilson et al. (1934) introduced an electrode of zero potential by connecting together the right arm, left arm and left leg electrodes through resistances of 5,000 ohms to a central terminal. This is called the neutral electrode. The exploring electrode, placed upon the chest wall at one of the positions mentioned above, will, therefore, record potential variations at that point. These unipolar leads are termed V leads (V standing for voltage) and so when the exploring electrode is in position 5 the resulting lead is designated V5.

If one wishes to record the potential variations in a limb this may be achieved by connecting the exploring electrode to that limb and pairing it with the neutral or indifferent electrode described above. These leads are designated VR, VL or VF according to whether the exploring electrode is connected to the right arm, left arm or left leg. The amplitude of the deflections in these leads

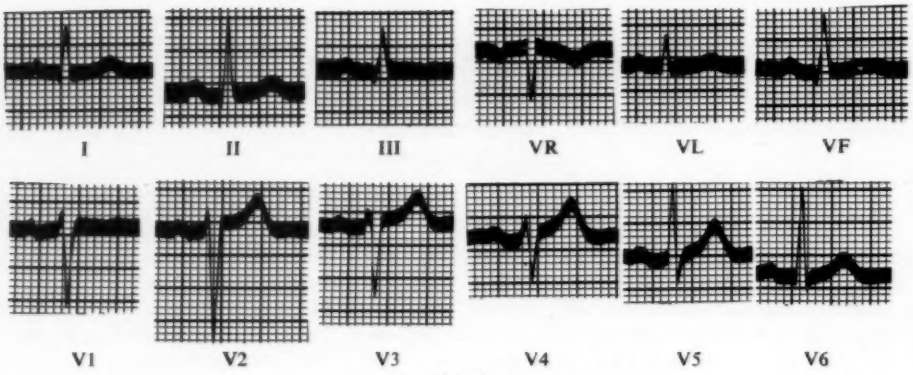

FIG. 1
Normal electrocardiogram.

is small and can be increased by 50 per cent, without altering the character of the waves, using Goldberger's (1942) modification in which the lead from the limb which is being explored is disconnected from the central terminal and the electrical resistances are removed. The resulting leads are termed augmented unipolar leads and are designated aVR, aVL, and aVF respectively. It is obviously impossible here to describe the various patterns which may be found in the unipolar limb leads in normal hearts; suffice it to say that they give vital information regarding the position of the heart.

It is customary to record routinely twelve leads, i.e., the standard limb, six precordial, and the unipolar limb leads. At the same time, multiple chest leads are at times unnecessary (as, for example, when one simply wishes to ascertain the nature of an arrhythmia), but when one suspects a myocardial infarct all the chest leads should be recorded.

THE NORMAL ELECTROCARDIOGRAM

The range of normality is wide, and, therefore, it will be as well to consider the normal electrocardiogram and discuss the variations which may be found. It is of paramount importance that the person who is interpreting the recording should have the fullest possible information regarding the history, clinical findings, therapy employed and the clinician's opinion of the case, for without this knowledge inaccurate conclusions may be drawn. Digitalis may produce various changes in the electrocardiogram which might be interpreted as indicative of serious cardiac changes if the interpreter is unaware that this drug is being prescribed. It is a good rule to examine each lead systematically so that no abnormality may be overlooked. The rate can be quickly estimated and note made of the rhythm, remembering that, in rare instances, an arrhythmia may be present in one lead only when a single-channel instrument is employed. The various components of the individual complexes are then studied in detail.

The P Wave. This is produced by the spread of the excitation process through the auricles from the S-A node. It is usually upright in leads I and II, but may be inverted in lead III, and even occasionally in lead II, while in the unipolar limb leads it is normally inverted in aVR but upright in aVF. In dextrocardia it is inverted in lead I. When the wave of excitation follows an abnormal pathway through the auricles the P wave will be deformed or inverted. This is observed in auricular or nodal premature contractions where one frequently finds the P wave to be inverted. In subjects with chronic pulmonary heart disease the P wave tends to be tall and pointed, up to 5 mm in height, while in left auricular hypertrophy resulting from mitral stenosis it is often bifid and widened (fig. 2).

P-R Interval. This is the time taken by the wave of excitation to travel from the S-A node to the ventricular muscle and is measured from the start

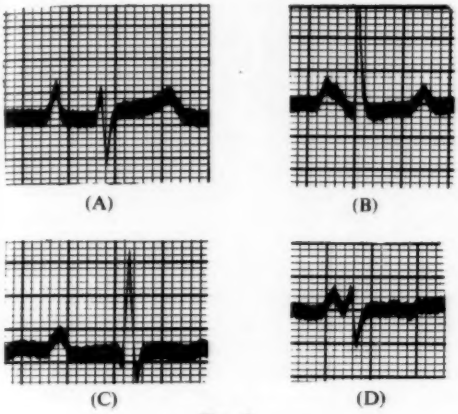

FIG. 2

(A) Tall pointed P wave in cor pulmonale.
(B) Wide bifid P wave in mitral valve lesion.
(C) Prolonged P-R interval, 0.28 second.
(D) Short P-R interval, 0.08 second.

of the P wave to the start of the QRS complex and ranges from 0.12 to 0.2 second. In young subjects and adults with slow heart rates this interval may be slightly longer, perhaps up to 0.22 second, but when the heart is rapid a prolonged P-R interval is almost certainly indicative of some delay in conduction. It may be short following the administration of atropine and also in the presence of A-V nodal premature contractions. Prolongation may result from active carditis, ischaemic fibrosis or digitalis therapy. Abnormal P-R intervals are seen in figure 2.

QRS Complex. This is produced by the spread of the excitation wave through the ventricular muscle. The first upward deflection in the complex is the R wave while the first downward deflection is the Q wave, provided it precedes the R wave. If, however, the first downward deflection follows the R it is called the S wave.

In the standard limb leads one notes the presence of axis deviation, remembering that this depends upon the position of the heart rather than the presence of ventricular hypertrophy. It is very important for one to be familiar with the QRS patterns normally obtained in the chest leads. In leads V1 and V2 the electrode lies over the right ventricle so that a right ventricular pattern is found consisting of a deep S and a small R wave. In leads V5 and V6, over the left ventricle, a tall R is seen, the S wave having disappeared. On moving the electrode across the chest from right to left a stage is reached where the change from right to left ventricular pattern occurs, called the transitional zone, which is normally found in lead V3 or V4 where the R and S waves are almost equal in amplitude. Change in the position of the heart will, therefore, alter the position of the transitional zone so that if the heart is rotated in a clockwise direction, as viewed from below, it will be displaced to the left, resulting in the appearance of a right ventricular pattern in leads V3 and V4. Conversely, anticlockwise rotation produces a left ventricular pattern in lead V3 or V2.

The width of the QRS complex is normally from 0.05 to 0.1 second. The amplitude of the deflections is important since a low voltage may indicate generalized myocardial change such as is found in myxoedema or constrictive pericarditis (fig. 10(c)). The presence of Q waves is extremely important, but it must be borne in mind that small Q waves may be present in leads I, aVL, aVF, V5 and V6 and deep Q waves in lead III in a perfectly normal heart.

RS-T Segment. This commences at the final deflection of the QRS complex and ends at the start of the T wave. Normally it lies on or very slightly above or below the iso-electric line. The shape of the S-T segment will be discussed later.

T Wave. This represents the process of recovery or repolarization in the ventricles and is upright in leads I and II but may be inverted in lead III. It may also be inverted in leads V1 and V2 in adults, but inversion in V3 is almost certainly abnormal except in children. An important feature of the normal T wave is that it is asymmetrical, so that if it be upright it has a slow ascent but a rapid descent, so that the apex is nearer to the finish than to the start of the wave.

Q-T Interval. This is measured from the start of the QRS complex to the end of the T wave and indicates the duration of ventricular systole. It is shortened by digitalis and prolonged in myocardial infarction, active rheumatic carditis, congestive failure and a reduced blood calcium or potassium level.

U Wave. This is a small upright wave which appears in the electrocardiogram after the T wave. Little is known regarding the production of this wave.

COMMON ELECTROCARDIOGRAPHIC ABNORMALITIES

A normal record does not exclude organic heart disease, so that a man with coronary atheroma, but without symptoms and with a normal recording, may be dead within a few hours from a myocardial infarct. On the other hand, a recording which appears to be grossly abnormal may not indicate serious heart disease. In figure 3 there is marked S-T segment depression with low amplitude T waves in a recording made on a man aged 38 who complained of chest pain which was not a typical cardiac pain, and on going into the history further it was discovered that he was receiving digitalis therapy. This was discontinued, and the second recording, taken six weeks later, reveals the S-T segment depression to have cleared and the T waves to be of normal amplitude.

VENTRICULAR HYPERTROPHY.

The diagnosis of ventricular hypertrophy is based mainly upon the findings in the chest leads and not upon the presence of axis deviation in the limb leads.

Left Ventricular Hypertrophy. The electrocardiogram may give the earliest indication of developing ventricular hypertropy. Since the ventricular wall is thicker than normal it will take longer for the impulse to pass through it and, therefore, the QRS interval will frequently be at the upper limit of normal or even, in the presence of marked hypertrophy, 0.11 to 0.12 second. In addition there is an increase in the potentials, so that in leads over the left ventricle a tall R wave is found, while over the right ventricle a deep S wave is present. Sokolow and Lyon (1949) found the voltage of the R wave in V5 and in aVL to be of considerable help in the diagnosis of left ventricular hypertrophy. In 20 per cent of those with hypertrophy of the left ventricle the voltage of R was greater than 26 mm in V5 and in 22 per cent it exceeded 10 mm in aVL. In severe cases S-T segment depression and even inversion of the T wave may develop in leads over the left ventricle, though it is felt that these indicate ventricular strain in addition to hypertrophy. The electrocardiographic changes are seen in figure 4 (B).

Right Ventricular Hypertrophy. The cardiographic changes in right ventricular hypertrophy vary considerably according to the degree of hypertrophy present. In moderate hypertrophy

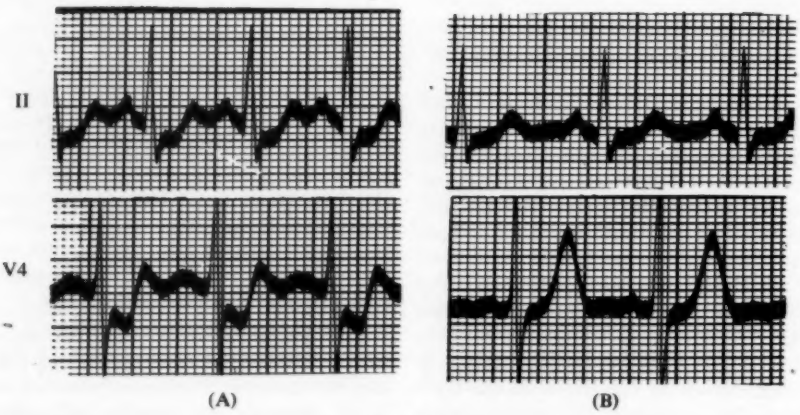

FIG. 3

(A) This shows a marked depression of the S-T segment due to digitalis therapy.
(B) Taken six weeks after digitalis has been discontinued. The S-T segment depression has disappeared and the T waves are of normal amplitude.

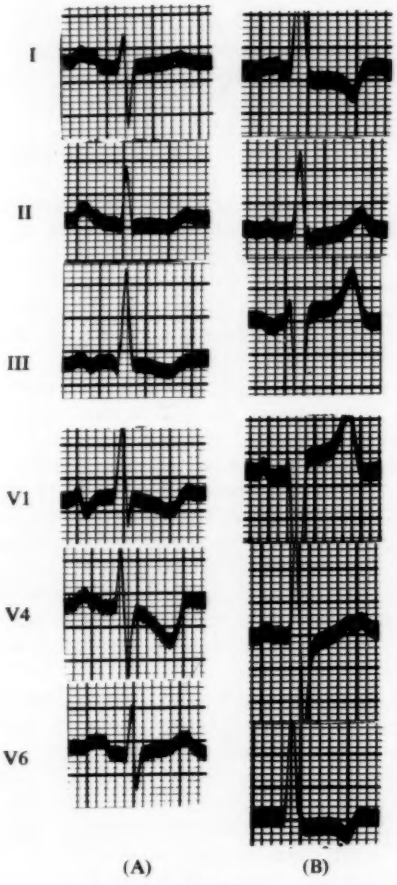

FIG. 4

(A) Right ventricular hypertrophy. Mitral stenosis.

(B) Left ventricular hypertrophy. Systemic hypertension.

right axis deviation is seen in the standard limb leads while in the chest leads deep S waves are found as far over to the left as lead V5. When a severe degree of hypertrophy and strain is present, tall R waves with S-T segment depression make their appearance in V1 and V2 with at times inversion of the T waves. The QRS interval may be slightly prolonged but this is not so marked as in the case of left ventricular hypertrophy. The changes may be seen in figure 4 (A).

Two conditions which may be confused with right ventricular hypertrophy are right bundle branch block and myocardial infarction and these will be discussed later.

CORONARY ARTERY DISEASE

As has already been mentioned, quite severe coronary artery disease may be present even when a normal or practically normal cardiogram has been obtained.

Myocardial Ischaemia. The cardiographic evidence of myocardial ischaemia varies from depression of the S-T segments to flattening or inversion of the T waves. In addition, there may be some delay in conduction resulting in slight prolongation of the QRS interval. In angina of effort, however, the electrocardiogram is frequently normal but proof of ischaemia may, in many instances be obtained by making a recording following exercise. In a healthy heart exercise will often produce minor changes in the recording consisting of minimal S-T segment depression, less than 0.5 mm, and a slight increase in the amplitude of the T waves. Twiss and Sokolow (1942) considered that in cases of angina the significant electrocardiographic changes after exercise are: S-T segment depression or elevation of 1.0 mm or more in lead I, of 1.5 mm or more in lead II, of 1.5 mm or more in lead III, and 2.0 mm or more in chest lead 4; or the conversion of an upright to a diphasic or inverted T wave in lead I, II or chest lead 4; or the development of a bundle branch block. The cardiogram is considered abnormal if any one of these changes appears. It must be stressed, however, that a normal record obtained after exercise does not exclude angina of effort. Master et al. (1944) considered the following changes in the electrocardiogram after the (Master) "two-step" exercise to be abnormal: a depression of the RST segment of more than 0.5 mm in any lead, a change from an upright T wave to an iso-electric or inverted T wave or T-wave changes in the opposite direction. In addition they carried out the 10 per cent anoxaemia test on each patient and found that the cardiographic changes corresponded almost exactly in both tests. They found that an upper respiratory infection, lung disease and fatigue may produce

abnormal results. A positive result is shown in figure 5.

It will be appreciated that the exercise test is not without danger and should be performed only when the diagnosis is in doubt.

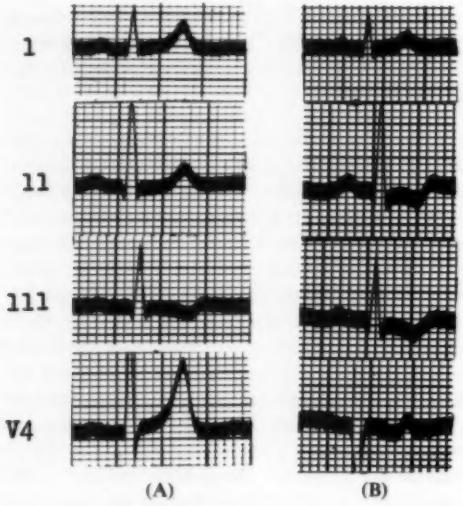

FIG. 5

Positive exercise test. (A) before exercising and (B) following exercise showing S-T segment and T wave changes.

Myocardial Infarction. The term infarction indicates that there is an area of necrosis in the heart wall, but this is not necessarily due to actual thrombosis within the coronary vessel, for it may result from a sudden fall in the blood pressure or from anoxaemia. Further, coronary occlusion may take place without infarction of the myocardium developing. The cardiographic changes of infarction usually become apparent within a few hours of the attack, but sometimes their appearance may be delayed for several days. If, therefore, the history is suggestive of infarction one must not be turned from this diagnosis by a normal electrocardiogram, and accordingly serial recordings should be made over the following few days. A normal record in itself will not, therefore, be necessarily helpful in attempting to differentiate between the acute pain of gall-bladder disease and a true cardiac pain. Moreover, severe upper abdominal pain may well be due to gall-bladder disease even though the cardiogram reveals evidence of an old myocardial infarct. The cardiogram must always be interpreted in the light of a considered clinical opinion.

The typical changes in a recent myocardial infarct are:

(1) The appearance of a deep and rather wide Q wave.

(2) Elevation of the S-T segment. This is seen as a high take-off by the S-T segment from the descending limb of the R wave. Further, there is a bowing of this segment, its convexity being upwards.

Over a period of a few days or weeks evolution of the infarct occurs, the S-T segment elevation becoming less marked, and, as it descends to the iso-electric line, the T wave becomes progressively more inverted. The main feature of the T wave is that it is symmetrical. With the passage of time the T wave shows a more shallow inversion and finally may become upright, but the Q wave frequently persists as evidence of the old infarct. Infrequently the S-T segment elevation persists and is then considered to be indicative of a ventricular aneurysm.

There are two main types of infarct, anterior and posterior. In an anterior infarct the typical changes appear in those chest leads which overlie the area of infarction, usually V3 to V6, while in the limb leads they are commonly present in leads I and aVL. The typical pattern is seen in figure 6. The standard limb leads, however, may remain normal or practically normal in an anterior infarct. If the changes are maximal in V3 to V4 or V5 to V6 the infarct is anteroseptal or anterolateral respectively.

In a posterior infarct the typical changes are present in leads III, aVF and sometimes, to a less marked degree, in lead II, while in lead I there is depression of the S-T segment. In the chest leads there is transient S-T segment depression. The typical changes of posterior infarction are shown in figure 7.

CONDUCTION DEFECTS

Bundle Branch Block.

This condition, which may be permanent, intermittent or transient, is due to a block in one of

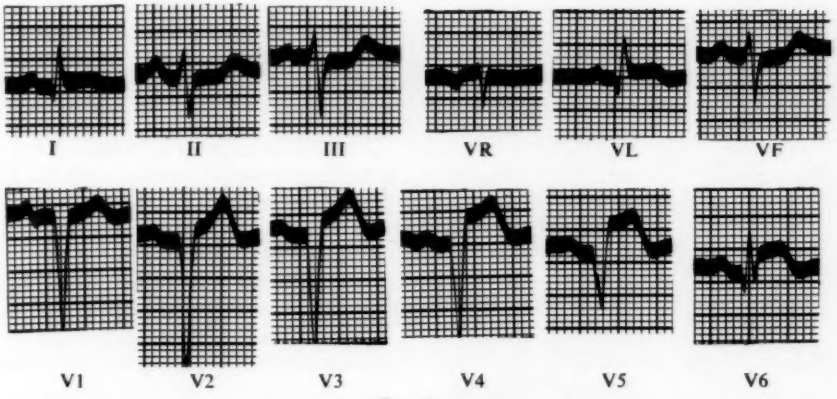

FIG. 6
Anterior infarct with S-T segment elevation in leads I, VL and the chest leads.

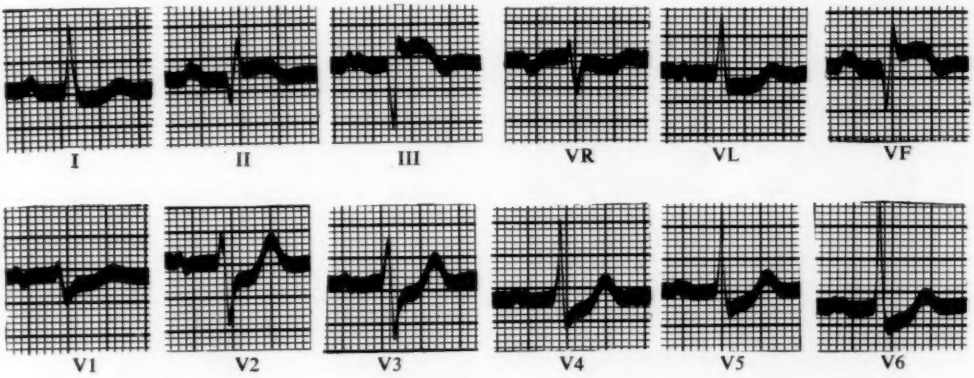

FIG. 7
Typical changes of a posterior infarct showing S-T segment elevation in leads II, III and VF, and depression in leads I and VL, and also in the chest leads.

the branches of the bundle of His. For example, in left bundle branch block the wave of excitation spreads in a normal fashion through the right ventricular muscle and thence to the interventricular septum and the left ventricle, and in consequence there is prolongation of the QRS complex. Figure 8(A) shows the changes which are found in left bundle branch block. In the standard limb leads a wide R wave with inversion of the T wave is seen in lead I, while in lead III the S wave is wide and the T wave upright. In V1 a small R wave is found with a deep S wave and slight upward displacement of the S-T segment, the T wave being upright. Over the left side in lead V6 there is S-T segment depression and inversion of the T wave but no Q wave.

In right bundle branch block the changes in the limb leads again depend upon the position of the heart, but usually there is a wide S wave in lead I with notching of the QRS complex. In V1 there is a small R with a tall R1, the T wave being inverted, while in V6 there is a wide S, small Q and an upright T wave. These changes can be seen in figure 8 (B).

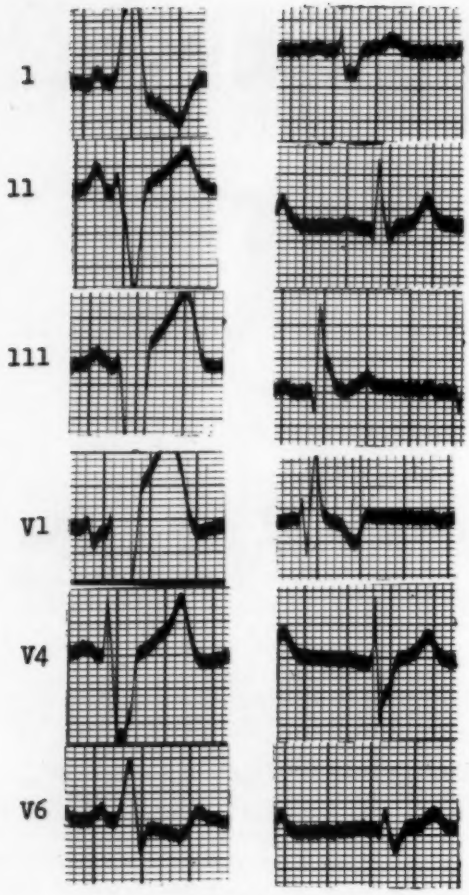

FIG. 8
(A) Left bundle branch block.
(B) Right bundle branch block.

Left bundle branch block is rather more common than right and the latter is sometimes found in quite young subjects who have no other evidence of cardiac disease.

Sino-auricular Block.

This is recognized by the absence of a complete complex in the electrocardiogram.

Auriculoventricular Block.

This varies from delay in conduction of the impulse from auricle to ventricle to complete heart block where no impulse passes from auricle to ventricle.

Partial heart block. (*a*) Prolonged P-R interval. Here there is simply delayed conduction between the auricle and ventricle (fig. 2 (C)).

(*b*) Wenckebach type of block. The P-R interval becomes progressively longer due to impaired conduction until finally the impulse fails to get through to the ventricles. This may be seen in figure 9 (A), where, at one stage in the record, the P wave is not followed by a QRS complex.

(*c*) Block with fixed A-V relationship. In this type the ventricle responds to every second, third or fourth auricular contraction. An example of 3:1 block is seen in figure 9 (B).

Complete heart block. No impulse from the auricle is transmitted through the A-V node so that the auricles and ventricles beat independently of each other. The P waves in the recording are regular, with a normal rate, while the QRS complexes are also regular but much slower. The stimulus for ventricular contraction arises in the A-V node, the bundle of His or in the ventricular muscle itself. When the pacemaker lies in the ventricular muscle the QRS complexes are widened as they are in ventricular premature contractions. An example of this type of block is shown in figure 9 (C).

PERICARDIAL DISEASE

Acute Pericarditis. Typically one finds elevation of the S-T segment, which has an upward concavity, in the limb leads and also frequently in the chest leads. The changes are due to damage to the superficial layer of the myocardium and are usually transient, lasting for a few days only, and then the S-T segment returns to the iso-electric line, though in some instances the T wave may become inverted. The changes of acute pericarditis may be confused with those of a recent myocardial infarct but may be differentiated by the absence of Q waves, the upward concavity of the S-T segment and the presence of S-T segment elevation in all limb leads in the former condition (fig. 10 (A)).

FIG. 9

(A) Wenckebach type of partial heart block
(B) 3:1 partial heart block.
(C) Complete heart block.

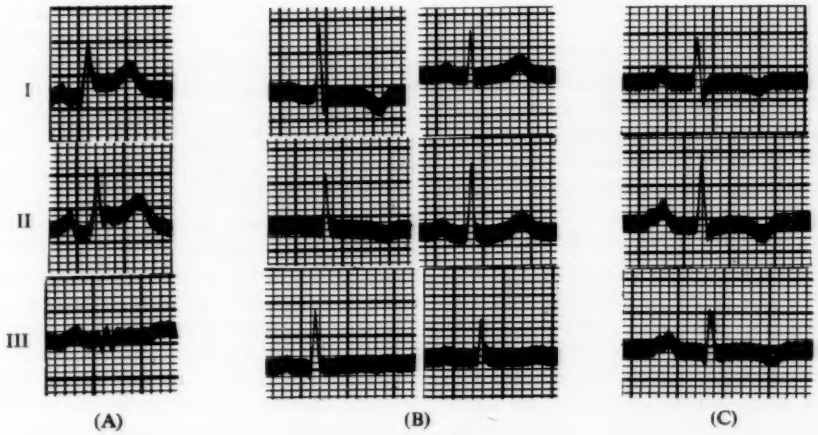

FIG. 10

(A) Acute pericarditis.
(B) Pericardial effusion before and after treatment.
(C) Constrictive pericarditis.

Pericardial Effusion. Fig. 10 (B) shows the changes present in a woman aged 34 years with a tuberculous pericardial effusion. The T waves are inverted but the voltage of the QRS complexes is usually rather lower than in the record shown. Following treatment, including aspiration of the fluid, the T waves have become upright.

Constrictive Pericarditis. The QRS complex is of low amplitude and the T wave flat or inverted, while in lead II the P wave is frequently bifid. This may be seen in figure 10 (C).

EFFECT OF DRUGS UPON THE ELECTROCARDIOGRAM

Digitalis. This tends to produce depression of the S-T segment, lowering of the T wave, shortening of the QT interval and prolongation of the P-R interval. The S-T segment depression, sagging in contour with an upward concavity, is best seen in leads II and V5. The effect of digitalis may persist for up to a month after it has been discontinued (see fig. 3).

Quinidine. This tends to increase both the P-R and QRS intervals in addition to producing depression of the S-T segment and lowering of the T wave. The S-T segment depression is not, however, so marked as it is with digitalis. From the practical standpoint the main effect of quinidine is its depressive action upon the S-A node and the conducting tissues of the heart.

ELECTROLYTE DISTURBANCES

Potassium. Hypopotassaemia produces depression of the S-T segment, prolongation of the Q-T interval, slurring of the QRS complex, flattening or inversion of the T wave and prominent U waves. Where the serum potassium level changes rapidly one may not have a true indication of the intracellular potassium level. Further, in hypopotassaemia other electrolyte changes may be playing a part in the production of the electrocardiographic appearances. Bellet et al. (1950) have described the electrocardiographic changes in 79 patients with hypopotassaemia. Henderson (1953) in a study of ten patients with diabetic acidosis found no correlation between the serum potassium level and the degree of electrocardiographic abnormality in 21 of the 42 abnormal recordings obtained. The Q-T interval could only be measured accurately in one instance and here the corrected time was normal. McAllen (1951) considers that the prolonged Q-T interval is due to the inclusion of the large U wave, so often present in hypopotassaemia. While the electrocardiogram in this condition has usually no characteristic form it shows, in some instances, an undulating outline produced by a depressed S-T segment, a low or inverted T wave and a high U wave. In hyperpotassaemia one finds tall pointed T waves.

Calcium. In hypocalcaemia the Q-T interval is prolonged due to lengthening of the RS-T interval, the T waves being normal.

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ELECTROCARDIOGRAPHY DURING ANAESTHESIA

BY

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INTRODUCTION

AS RESEARCH in anaesthesia develops, the attention of the anaesthetist is being directed more and more to a closer study of the cardiovascular system under the varying conditions of surgery and anaesthesia. The immediate success of all surgical and anaesthetic procedures depends primarily on the patient's cardiovascular system and its ability to withstand the direct and indirect stresses to which it is subjected by the surgeon and the anaesthetist. This would appear to be a self-evident fact, yet, in the assessment of new techniques and manoeuvres, it is surprising how often it has been overlooked. The past few years have seen the introduction of several new and revolutionary anaesthetic techniques; the published reports of these often contain only the briefest and most indefinite references to the cardiovascular states of the patients before, during, and after anaesthesia.

In attempting to assess the value of an anaesthetic technique it is advisable to apply a system of measurement which is reasonably accurate and acceptable to every anaesthetist. In the estimation of cardiovascular function during anaesthesia, and indeed in the estimation of practically every vital function, clinicians have a restricted choice of instruments which can be conveniently used for obtaining accurate records. Pulse palpation can be inaccurate and misleading, and blood pressure estimations are not a reliable index of myocardial function. The electrocardiogram provides us with a record of the heart rate, its rhythm, the site and number of the pacemakers, the efficiency of the conducting tissue, and, to a certain extent, it may depict changes in the position and size of the heart. The electrocardiogram also provides a convenient method for recording fluctuations of autonomic tone occasioned by the numerous drugs now at our disposal, and it is a valuable index of the electrolytic balance of the blood; it is indispensable in deciding what is the cause of cardiovascular collapse in the anaesthetized patient (Johnstone, 1955).

The electrocardiogram is essentially a picture of the site of origin of the stimulus potential and the speed and direction in which it travels to initiate the myocardial contraction—in other words, it depicts the activity of the Purkinje fibres. It provides no indication of the strength of the myocardial contraction. The Purkinje conducting tissue and the myocardium derive nutriment from a common blood supply—the coronary arteries; therefore, if it is observed that certain drugs or manoeuvres are causing electrocardiographic evidence of impairment of conductivity, I feel it is reasonable to assume that the myocardium is involved in the deterioration. The classic signs of diminished conductivity are widening of the QRS complex with displacement of the ST segment and alterations in the shape of the T wave. At this stage in the development of the electrocardiology of anaesthesia I do not propose to enlarge on these changes, as our knowledge is by no means complete.

EQUIPMENT

The most suitable type of electrocardiographic equipment for theatre use is the valve amplifier which combines a direct-writing galvanometer monitored by a cathode ray oscilloscope. The galvanometer and the monitor circuits should be wired in parallel to enable them to be used independently: the direct writer is used intermittently for control checks and for obtaining permanent records of important changes, whilst the monitor is used continuously throughout the operation. Electrodes and leads are positioned so

as not to interfere with the operation site, and, when single lead electrocardiography is in use, it is generally advisable to attach a third electrode and lead to the patient to " earth " the currents induced from neighbouring electrical equipment.

The recording galvanometer should be sensitized according to the Einthoven convention of 1 cm deflection for 1 millivolt. The amplitude of the deflections on the monitor is adjusted to any sensitivity which permits easy observation when it is placed in a relatively remote part of the theatre.

RECOGNITION OF EXTRACARDIAC POTENTIALS (ARTEFACT)

It has long been known that fallacies may arise in the interpretation of electrocardiograms taken during surgical operations: fibrillary and ectopic auricular contractions are closely simulated by tremors in skeletal muscle; large steel retractors may induce static discharges or may short-circuit the cardiac potentials; and interference from electrical equipment is also a frequent source of disturbance.

Figure 1 illustrates a series of electrocardiograms distorted by various types of extracardiac interference:

A shows the superimposition of the 50-cycle alternating current (AC) interference on the cardiogram and its elimination by " earthing " the patient. Usually this interference is eliminated by the " earth " terminal on the power plug and it is seldom necessary to use a separate " earth " lead. Endoscopes in situ are the worst offenders in this respect, and AC interference from them is very difficult to eliminate. It is still possible to identify the cardiac potentials through the AC " hum."

B illustrates the effect of diathermy on the cardiogram. This interference is impossible to eliminate and it completely obliterates the cardiac potentials. It does not constitute a serious drawback as its use is transient. The diathermy current does not damage the amplifying or monitoring circuits, but its high frequency vibrations may shatter the galvanometer pen; for this reason it is advisable to have the galvanometer on an independent circuit.

C shows interference due to tense somatic muscles in a patient awaiting the induction of anaesthesia. The P waves have been obscured and it is impossible to determine the site of the pacemaker.

D shows, in the same patient as C, the abolition of somatic interference by the administration of thiopentone. The P waves become clear as soon as the patient loses consciousness.

E illustrates interference encountered when scissors—metal friction—or large retractors are used during the operation. These deflections

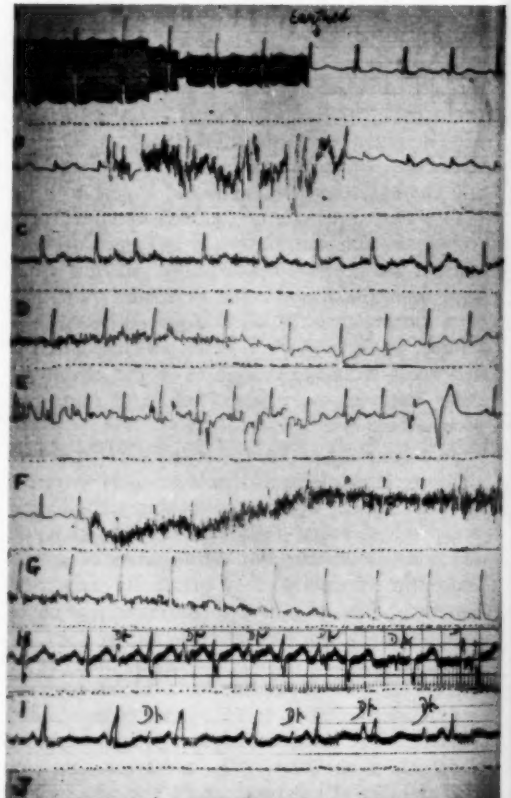

FIG. 1

Cardiograms illustrating extracardiac interference. For detailed explanations see text.

often closely simulate ectopic contractions (extrasystoles) and activate certain types of electronic cardiotachometers, causing these instruments to give erroneously high recordings of the pulse rate.

F shows somatic interference caused by the intravenous administration of suxamethonium.

G shows the disappearance of the somatic potentials with the onset of suxamethonium paralysis.

H was taken during trichlorethylene tachypnoea. The deflections marked "Dp" are due to diaphragmatic contractions and closely resemble auricular ectopic beats.

I shows distortion of the cardiogram by hiccoughing during upper abdominal surgery in an inadequately anaesthetized adult.

J shows considerable alternations in the electrical potential of the cardiac deflections during anaesthesia in a patient under controlled respiration. During pulmonary inflation the R wave becomes very small. This diminution of voltage suggests a change in the position of the heart relative to the skin electrodes and does not indicate myocardial deterioration. Change in the voltage of the cardiac deflections (electrical axis) may be induced by the Trendelenburg and lateral positions, by complete curarization, and by thoracotomy. These factors should be taken into account when assessing the effects of drugs or operations on the cardiogram.

ELECTROCARDIOGRAPHIC ABNORMALITIES DUE TO AUTONOMIC IMBALANCE

The commonest electrocardiographic abnormality encountered in the anaesthetized patient is a change either in the position or number of the pacemakers. Widening of the QRS complex, shift of the ST segment, and alterations in the T wave have not been fully investigated and their significance is not yet clear. Pacemaker changes, however, have been well documented and their significance is more fully understood; this paper will be concerned mainly with those abnormalities which, depending on the aetiology, fall into three main groups: those due to vagal activity, those secondary to sympathetic stimulation, and the fibrillary phenomena.

Figure 2 illustrates diagrammatically the myocardium with its conducting system and autonomic innervation; superimposed are examples of cardiograms obtained from the particular areas of the heart to which they are related on the diagram. The sinus node and the atrioventricular node are richly innervated by both sympathetic and parasympathetic fibres and their activity is governed by these nerves, the sympathetic being stimulant and the parasympathetic inhibitory. The ventricular myocardium and the peripheral Purkinje network have a well defined sympathetic innervation but the parasympathetic supply of these structures has not been demonstrated anatomically, though its influence can be observed by physiological means (Johnstone, 1953).

ELECTROCARDIOGRAPHIC DISTURBANCES DUE TO VAGAL STIMULATION

Increase in vagal tone may be induced by a variety of methods in nonatropinized patients: inhalation of irritant anaesthetic vapours may cause reflex vagal inhibition of the heart (pulmocardiac reflex); administration of parasympathetic stimulants such as neostigmine or suxamethonium; high spinal anaesthesia by abolishing the normal sympathetic countereffect; by surgical stimulation either of the parasympathetic centre in the hypothalamus or of the vagus nerve peripherally; and sometimes by overdistension of the lungs during controlled respiration.

Vagal stimulation causes slowing of the sinus rate with flattening of the P wave (fig. 2A and 2B). More intense stimulation may inactivate the sinus node causing cardiac arrest (fig. 2P). The efficiency of the atrioventricular node and the bundle of His may be impaired by vagal stimulation, leading to partial heart block (fig. 2G: note prolonged PR interval), or to complete heart block with ventricular standstill (fig. 2H); rarely the vagal stimulus may block one of the branches of the bundle of His, producing the electrocardiogram typical of intraventricular (bundle-branch) block (fig. 2R).

More often, however, when the sinus node is inactivated by vagal stimulation, the atrioventricular node escapes and takes over the role of pacemaker with the appearance of characteristic electrocardiograms, the form of which is related

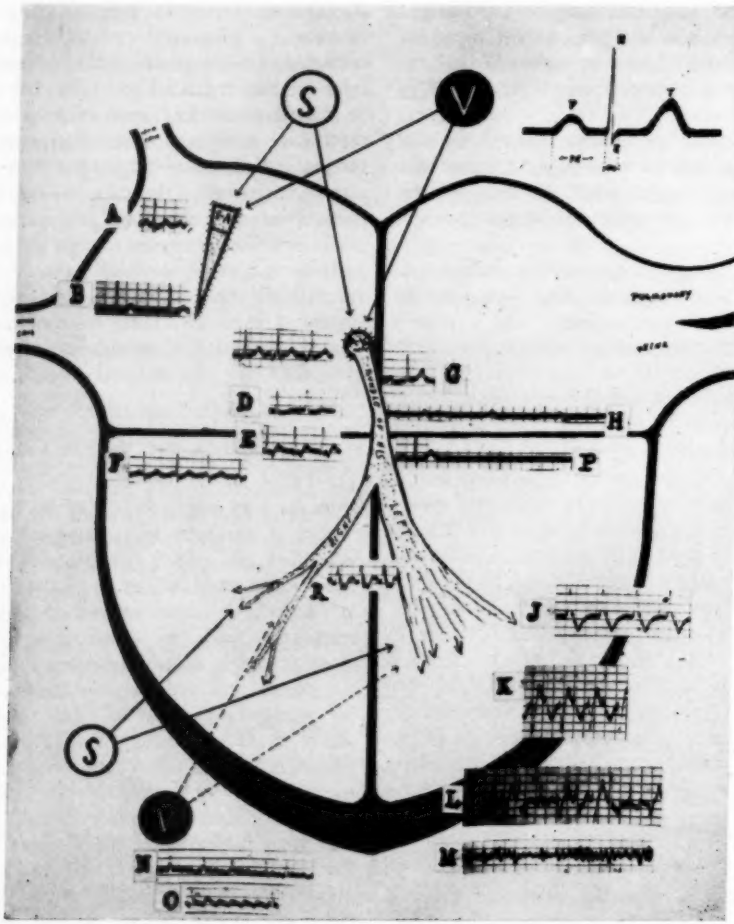

FIG. 2
Diagram of the myocardium, the Purkinje tissue, and the autonomic innervation. See text for explanation of cardiograms.

M. Ventricular fibrillation.
N. Auricular fibrillation.
O. Auricular flutter.
S. Sympathetic innervation.
V. Parasympathetic innervation.

to the site of the pacemaker within the atrioventricular node and bundle. When the pacemaker is within the atrioventricular node the stimulus spreads backward to the auricles (retrograde spread) and an inverted P wave precedes the ventricular complex (fig. 2C); a pacemaker within the bundle causes synchronous contractions of the auricle and ventricle, the P wave being buried in the ventricular complex and therefore invisible (fig. 2D); a pacemaker situated low in the bundle near its bifurcation causes ventricular contraction to precede the auricular contraction, when an inverted P wave follows the R wave (fig. 2E). Vagal stimulation not

infrequently causes the auricles and ventricles to beat independently (atrioventricular dissociation), the auricles being controlled by the sinus node and the ventricles by the atrioventricular node; sometimes an occasional sinus impulse will be conducted to the ventricles, causing a pulse irregularity, referred to as atrioventricular dissociation with interference (fig. 3C).

ELECTROCARDIOGRAPHIC DISTURBANCES DUE TO SYMPATHETIC OVERACTIVITY

The effects of sympathetic stimulation are seen mainly in the sinus node and in the peripheral parts of the Purkinje network and are essentially those of increased irritability of the cells in these structures. Moderate stimulation causes sinus tachycardia with increase in the amplitude of the P wave and the appearance of occasional ventricular extrasystoles, due to activation of cells in the ventricular portions of the Purkinje system. More intense stimulation produces more frequent ventricular extrasystoles which may eventually dominate the cardiac activity, either in the form of a multifocal ventricular tachycardia (fig. 2L), bifocal ventricular tachycardia (fig. 2K), or a unifocal ventricular rhythm (fig. 2J). Note the similarity in the electrocardiographic form of a unifocal rhythm and an intraventricular block.

Sympathetic overactivity may be precipitated by a variety of factors:

(1) The induction of anaesthesia by the inhalation of gases or vapours, particularly if it is associated with struggling and partial asphyxia.

(2) The use of agents which are known to activate the sympathetico-adrenal mechanisms, e.g. chloroform, cyclopropane and trichlorethylene.

(3) By the administration of adrenaline or noradrenaline.

(4) By the retention of carbon dioxide which has pharmacological properties not unlike those of adrenaline.

(5) By the surgical manipulation of suprarenal tumours.

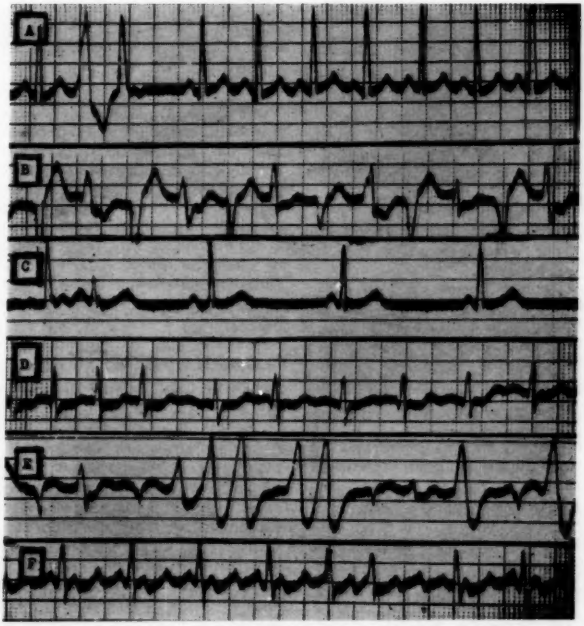

FIG. 3
The irregular pulse during anaesthesia. See text for explanation.

The hypertensive effect of sympathetic stimulation may produce reflex vagal inhibition of the heart through activation of the baroceptors in the aortic arch and carotid sinuses; the true effects of sympathetic stimulation on the heart will therefore be rendered more obvious by the administration of atropine.

THE IRREGULAR PULSE DURING ANAESTHESIA

The nature of a pulse irregularity is best determined by means of the electrocardiograph. Some of the causes of the completely irregular pulse are illustrated on figure 3; sinus arrhythmia and the rarer forms of sinus block are not included. Tracing 3A shows a sinus rhythm interrupted by isolated ventricular extrasystoles, and tracing 3B shows a multifocal ventricular rhythm in which the sinus beats have been completely replaced by ventricular extrasystoles.

Tracing 3C illustrates an atrioventricular dissociation with interference. This abnormality is invariably due to vagal overactivity and is best treated by the intravenous administration of atropine.

Tracing 3F is one of auricular flutter with an irregular ventricular response. This is an uncommon complication of anaesthesia but it occurs in experimental work on auricular fibrillation in which auricular flutter is seen during the transition from sinus rhythm to auricular fibrillation. Tracing 3D is of auricular fibrillation. Tracing 3E is of auricular fibrillation with frequent ventricular extrasystoles.

THE FIBRILLARY PHENOMENA

Fibrillation, which may affect either the auricles or the ventricles, does not appear to be directly related to autonomic imbalance as are the other forms of arrhythmia encountered during anaesthesia. Important work on the aetiology of auricular fibrillation has recently been reported by Professor Burn and his colleagues (1956). It would appear that in fibrillation the abnormality lies in the myocardial cells rather than in the cells of the conducting tissue.

Ventricular fibrillation is often alleged to be the cause of cardiovascular collapse during anaesthesia, but unfortunately the exact circumstances in which it occurs and its electrocardiographic confirmation are seldom presented. As yet, comparatively little is known of its aetiology, though it is almost certain that it does not occur without preceding electrocardiographic changes. There is little doubt that it may be caused by several factors, all of which probably have myocardial ischaemia as a common predisposing effect. Every effort should be made to identify the predisposing causes and I feel sure that it will be much more satisfactory to prevent the occurrence of ventricular fibrillation than to devise complex and dangerous methods of treatment for it.

Auricular fibrillation is a rare complication of anaesthesia although frequently encountered in the pre-operative and postoperative phases. In most cases established auricular fibrillation is uninfluenced by anaesthesia, but the atrioventricular node, the bundle of His, and the peripheral Purkinje network are still subject to autonomic effects: figure 3E shows a pre-operative auricular fibrillation with frequent ventricular extrasystoles precipitated by the induction of anaesthesia with trichlorethylene.

Auricular fibrillation may sometimes be abolished by general anaesthesia, as the following illustrates.

CASE 1. Male, 72 years. Hemicolectomy for carcinoma of the colon. Hypertensive heart disease with auricular fibrillation of at least six weeks duration. Premedicated with atropine 0.5 mg. Anaesthesia induced with thiopentone 250 mg and maintained with cyclopropane and oxygen in a closed circuit with assisted respiration. After twenty minutes of cyclopropane anaesthesia the auricular fibrillation reverted to auricular flutter (fig. 4C) and five minutes later a sinus rhythm was present (fig. 4D). Sinus rhythm persisted throughout the remainder of the operation and during convalescence.

In rare instances patients subject to paroxysmal auricular fibrillation may develop the arrhythmia at any stage of the operation.

CASE 2. Male, 63 years. For suprapubic prostatectomy. Fit and well. Heart and lungs normal. History of severe palpitations precipitated by emotional stress; each attack of palpitations lasted several hours. Premedicated with atropine 0.6 mg and pethidine 50 mg. On arrival at the operating theatre a sinus rhythm was present (fig. 5A). A few minutes later, whilst a lumbar puncture was being performed for the induction of spinal analgesia, auricular fibrillation suddenly developed (fig. 5B). 1.2 ml of heavy cinchocaine were injected into the subarachnoid space and the patient placed in about 10 degrees of Trendelenburg position. Five minutes later spinal analgesia was present and

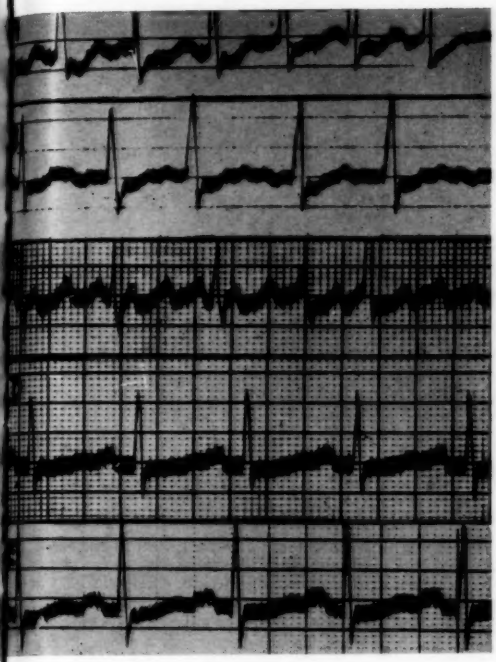

FIG. 4

Case 1. All lead 2. Male, 72 years. Hemicolectomy.
A. Before induction. Auricular fibrillation.
B. After thiopentone. Auricular fibrillation.
C. After cyclopropane for 20 minutes. Auricular flutter.
D. After cyclopropane for 25 minutes. Sinus rhythm.
E. During recovery of consciousness.

the systolic pressure had dropped to 90 mm Hg; auricular fibrillation was still present. Thiopentone 250 mg were injected intravenously to eliminate any emotional factors which may have been related to the fibrillation; the systolic pressure then dropped to 60 mm Hg, the fibrillation remained unchanged and the ventricular response rose to 160 beats a minute (fig. 5C). Methoxamine, 10 mg intravenously, restored the systolic pressure to 120 mm Hg, but failed to influence the fibrillation. Digoxin 0.5 mg was then injected intravenously and sinus rhythm returned five minutes later (fig. 5D). A suprapubic prostatectomy was performed without difficulty and the patient made an uneventful recovery without any further paroxysms of auricular fibrillation.

These tracings are presented in order to illustrate the use of electrocardiography in determining the cause of a pulse irregularity; without this information it would be difficult to select the specific therapy required for the correction of the irregularity. Regularity of the pulse does not imply normality of the rhythm, for ventricular extrasystoles may occur when the ventricle is empty, and therefore fail to produce a palpable pulse, but nevertheless block the following sinus beats and cause what appears on palpation to be a bradycardia. This point is illustrated in the following example.

CASE 3. Female, 54 years. Poorly nourished. Six months history of loss of weight, increasing lassitude and weariness, loss of appetite, indigestion, and

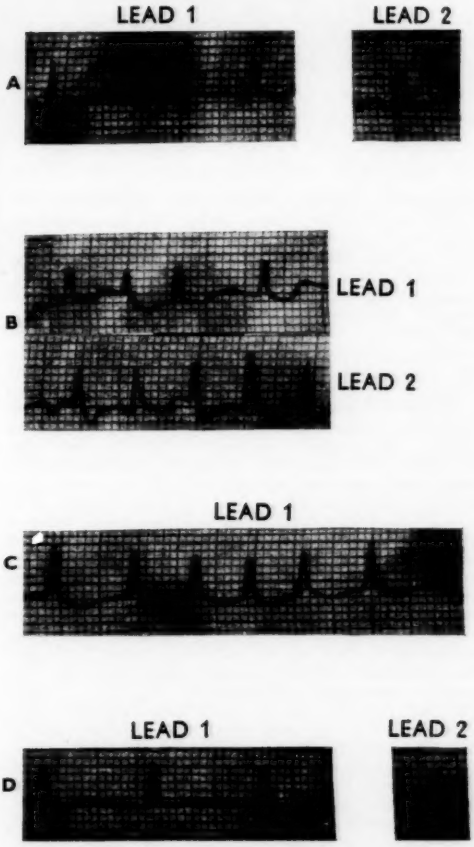

FIG. 5
Case 2. Male, 62 years. Prostatectomy.
See text for explanation.

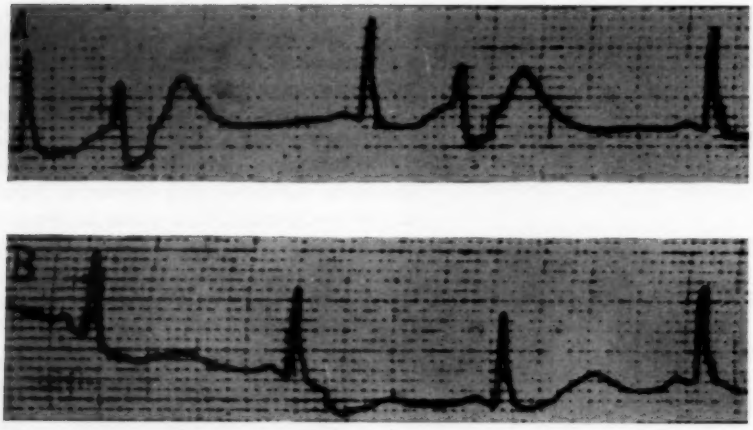

FIG. 6
Case 3. Female, 54 years. Laparotomy. All lead 2. See text for explanation.

occasional vomiting. Radiological examination revealed a doubtful filling defect in the stomach, necessitating an exploratory laparotomy. Premedication was with atropine 0.6 mg and pethidine 50 mg. Anaesthesia was induced with 400 mg of thiopentone, an endotracheal tube inserted and a nitrous oxide-oxygen mixture administered. Prior to opening the peritoneal cavity 20 mg of laudexium methylsulphate (Laudolissin) were injected intravenously and respiration controlled. Nothing abnormal was found in the gastro-intestinal tract and the abdomen was closed. Neostigmine 2.5 mg with atropine 1 mg were then administered intravenously, but failed to restore spontaneous respiration. During the next twenty minutes a further 7.5 mg of neostigmine were given with atropine 0.5 mg to keep the pulse rate within reasonable limits, but complete apnoea persisted. Controlled respiration was continued, using a 50 per cent oxygen-nitrous oxide mixture with a total gas flow of 10 litres per minute from a Boyle's apparatus. Thirty minutes later the patient was found to be in a collapsed state, pale, sweating, radial pulse rate 40 per minute, systolic pressure 65 mm Hg, and still completely paralysed; a cholinergic crisis was suspected in view of the very slow radial pulse and the sweating. An electrocardiogram, however, revealed ventricular extrasystoles (fig. 6A). Carbon dioxide retention due to inadequate ventilation was suspected. Vigorous ventilatory efforts, with about 20 litres per minute from the Boyle's machine, failed to abolish the arrhythmia and the blood pressure dropped still lower. The endotracheal tube was removed and a cuffed tube reinserted and the patient ventilated with a Coxeter-Mushin circuit with soda lime and a 10-litre-per-minute gas flow. Three minutes later the extrasystoles disappeared and a steady sinus rhythm of 75 beats per minute returned (fig. 6B): systolic pressure rose to 85 mm Hg and the sweating ceased. Seven hours later spontaneous respiration returned and the patient made an uneventful recovery. Postoperative investigations revealed the presence of myasthenia gravis, affecting mainly the muscles of the upper limbs; neostigmine therapy abolished the patient's symptoms.

CONCLUSION

This paper by no means covers the many aspects of the electrocardiology of anaesthesia. The subject is at an early stage of development, but it is becoming more useful as an aid in reducing the risks of surgery and anaesthesia. It is a subject which can best be appreciated by practical experience, as the range of both normal and abnormal cardiograms is wide. The significance of electrocardiography has been ably described in the words of David Scherf and Adolph Schott (1953 a, b): "At first sight this topic may appear to be a very limited one, but closer study proves the view to be erroneous and reveals not only its considerable scope and complexity but also the numerous links which relate it to other biological problems, physiological as well as clinical."

ACKNOWLEDGMENTS

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SOCIETY NEWS

GLASGOW AND WEST OF SCOTLAND SOCIETY OF ANAESTHETISTS

On Saturday, October 20, 1956, at Adam House, Edinburgh, a Joint Meeting was held with the Association of Anaesthetists of Edinburgh. Dr. McCallum Miller, of Edinburgh, presided, and the speaker was Dr. W. N. Rollason, of Hull. The subject was "The Role of the Anaesthetist in the Treatment of Facial Neuralgias."

Dr. Rollason said that the treatment of facial neuralgias involved dentist, neurologist, neurosurgeon, and anaesthetist whose place in treatment was increasing in importance. The speaker at this point paid tribute to Wilfred Harris for his work on this subject.

The patients are referred to the Anaesthetic Outpatient Department, and to date number 41, of whom 26 were female and 15 male.

Facial neuralgias are divided into the following types: dental, ocular, auricular, atypical, postherpetic, glossopharyngeal, tic douloureux, intramedullary, osteitis, tumour, and psychogenic. The diagnosis is made from the history and physical examination with X-ray investigation of skull, sinuses, temperomandibular joints, cervical spine and teeth. Blood is taken for Wasserman and Khan reactions and urea estimation. Urine analysis is done.

When the diagnosis is made, the specific type of facial neuralgia is referred to the appropriate specialist, the commonest cause being dental.

Tic douloureux occurs usually in people over 50 years of age. The constant symptom is pain—unilateral, trigeminal in distribution, agonizing, paroxysmal, precipitated by an event such as shaving, talking or washing. Certain trigger zones are present where light touch to a particular area will precipitate tic douloureux. Facial analgesia and absence of the corneal reflex are never found. The differential diagnosis is stressed as being of the greatest importance. Treatment is under the control of the specialist most concerned. The auricular type may be helped by injection of the great auricular nerve; the postherpetic by X-ray therapy; glossopharyngeal by topical anaesthesia to the areas or by nerve block in the neck. The intramedullary type is due usually to syringomyllia and is treated by analgesic drugs and chlorpromazine. The neoplastic type is usually accompanied by paralysis and anaesthesia and is referred to the neurosurgeon, while neuralgia due to psychogenic causes is precipitated by emotional trauma and may respond to psychotherapy.

Tic douloureux in the early stages may be controlled by phenobarbitone, pure trichlorethylene, nicotinic acid, radiotherapy, vitamin B12. Before commencing radical treatment its effects must be plainly explained to the patient who must elect to undergo it. The points to be stressed are that the numbness will be permanent and that the eye on the affected side may be involved.

Dr. Rollason considered that in young patients operation is the procedure of choice, injection being reserved for the elderly. With injection the the death rate is nil, the failure rate high, and the technique difficult. Thirty-five cases have been injected using the anterior approach. The method of injection was described and the precautions taken during and after treatment, along with an impressive list of possible complications.

In conclusion Dr. Rollason described the conditions in a provincial hospital without readily obtainable neurosurgical aid, and stressed the necessity of accurate diagnosis.

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THE EVOLUTION OF ANAESTHESIA

BY

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(*Continued from Vol.* 28, *page* 283)

THE history of anaesthesia is a large subject; paradoxically, it is also true that it does not exist at all. Anaesthesia is merely a branch of medicine, and its history can only be understood by examining the whole subject. Equally, medicine is a branch of science, while science is but one facet of human activity; it follows, therefore, that the history of anaesthesia can only be interpreted correctly by viewing it in the perspective of other human activities, medical, scientific and social; inventions, discoveries, wars, religious beliefs, and all the manifestations of art and culture.

C. *The Scientific Age* (585 B.C.–A.D. 200).

The prediction by Thales of an eclipse of the sun in 585 B.C. marks the beginning of the rise of Greek science, which, in medicine, was to come to an end with the death of Galen. The attitude of the Greek philosophers to life in general was quickly reflected in the physician's attitude to disease: the natural history of disease was studied and rational treatment was instituted. Because the causation and nature of disease were often misunderstood, the treatment was frequently incorrect, although it was nevertheless rational.

Parallel with the treatment of disease by physicians, magical treatment by priests was also carried on in temples erected for the worship of Asklepios. Here, again, treatment became more rational, employing baths, physiotherapy and some degree of "mental rehabilitation", but the essentially magical nature of treatment in the Asklepieia must not be forgotten. The final defeat of the Persians ensured the success of rational medicine in its fight against superstition.

Greek medicine reached its first peak in the time of Hippocrates of Cos, and much evidence of its modernity in outlook is to be found in the Hippocratic writings, the majority, if not all, of which were written by members of the "Hippocratic" school; they certainly reflect the teachings of the "Father of Medicine". From Asia Minor Greek medicine spread to, and flourished in, Athens during the Golden Age of Pericles, which ended with the plague and the Spartan victory. Later the conquests of Alexander the Great, whose father had brought all Greece under his rule, led to the founding of Alexandria, with its "University". Here Greek medicine reached its second peak with Erasistratus of Chios and Herophilus of Chalcedon. Hitherto, respect for the dead had prevented human dissection, although animals had been frequently examined. Now, for the first time, human anatomy was closely inspected.

The conquest of Greece, and later Egypt, by the Romans, shifted the medical centre to Rome, but the light of Alexandria still burned, although not so brightly as of yore. The interest of Rome in war and conquest attracted those with medical leanings to the post of military surgeon, a craft which, even in those days, was considered separate from, and of a lower social status than that of physician. The physicians were mainly Greeks, and wrote in that language (e.g. Asklepiades and Galen). The only medical works in Latin which have come down to us are the *De re medicina* of Celsus and the *Historia Naturalis* of Pliny, both written in the 1st century A.D. It is here that mandragora makes its appearance in medical literature.

Galen was the last great physician of the scientific era, and this was in part his own fault. A prolific author, he wrote more than 100 books, and he influenced posterity to a greater degree than any other medical man. His dogmatism and the teleology of his ideas would have smothered scientific spirit in suitable surroundings: the spreading of Christianity was not favourable to

science, while, in Arabia, Islam also resisted scientific advance. Thus Galen remained the supreme authority until the Renaissance.

Greeks and Romans alike seem to have had no knowledge of anaesthesia, although the soothing and pain-relieving attributes of opium were appreciated certainly in the 4th century B.C., and probably earlier. Belief in the narcotic power of mandrake sprang up towards the end of the scientific era, while opium seems to have been less and less used. Mandragora will, however, be dealt with in the next section, where it logically belongs.

B.C.

c. 624–565. Thales of Miletus (see above).

c. 580–500. Pythagoras, probable originator of the doctrine of four elements and four qualities, later applied to medicine. The four humours, Blood (like Fire, hot and dry), Phlegm (like Water, cold and wet), Yellow Bile (like Air, hot and wet), and Black Bile (like Earth, dry and cold), regulated the action of the body, and an upset in the proper proportions of these humours was a cause of disease. Later, therefore, herbals listed drugs as being "dry and cold", or "wet" and so on "to such-and-such a degree", to indicate the sort of complaint for which they would be suitable. The theory received much support from Aristotle.

509. Expulsion of the Tarquins from Rome and the foundation of the Roman Republic.

496–406. Sophocles, the great tragedian.

490. Battle of Marathon: the Persian army is defeated by the Greeks under Miltiades.

480. Battle of Salamis: the great naval battle in which the fleet of Xerxes was destroyed by the Greeks.

c. 480–425. Herodotus, the first historian. His history of the struggle between Greece and Persia has been mentioned above.

469–399. Socrates. According to Xenophon, Socrates was aware of the sedative action of opium.

c. 460. Birth of Hippocrates on the island of Cos. His name survives in the "Facies Hippocratica" and in the "Hippocratic Oath", which still remains the foundation of the code of medical ethics. The celebrated aphorisms contain the famous saying, "Life is short, and the Art is long, opportunity fleeting, experiment dangerous and judgment difficult." He lived to old age.

c. 450. The time of Nehemiah in Jerusalem. About this time the Hexateuch (i.e. the Pentateuch and the Book of Johsua) was compiled from earlier documents. The account of the birth of Eve (Genesis ii, 21) has been used as evidence of anaesthesia at an early date. It seems much more likely, however, that the sleep which was cast upon Adam was a device to keep God's secret rather than that

B.C.

the human chronicler had any idea of surgical anaesthesia. There is no evidence that the Israelites used any form of pain-relief.

The Hebrew word "dodaim", translated as "mandrakes", occurs also (Genesis xxx, 14–17), but only with magical significance in connexion with fertility.

443–429. Pericles rules Athens, the "Golden Age".

427–348. Plato, disciple of Socrates, and founder of the Academy; his views on the soul and on the universe led to the belief that the body (microcosm) and the heavens (macrocosm) were interdependent, and so supported the application of astrology to medicine.

404. Athens falls to Sparta.

384–322. Aristotle; in about 342 he became tutor to Alexander the Great, then about 14 years old. He made great discoveries in comparative anatomy and embryology, studying the development of the chicken and dissecting and describing the natural history of many animals and fish. Although he had no comprehension of evolution, his "Ladder of Nature", in which he arranged plants and animals in an ascending scale of complexity, was the first step towards this idea.

382–322. Demosthenes, Athenian orator and statesman. In one of his speeches he referred to the lethargy of the Athenians as resembling the sleep induced by mandragora or some other narcotic.

c. 371–287. Theophrastus, pupil and friend of Aristotle, sometimes called the "Father of Botany", described the poppy, and also mentions mandragora.

323. Death of Alexander the Great; the empire which he created fell to pieces on his death, the Seleucids obtaining the East, and the Ptolemies, Egypt. Ptolemy I Soter began the great library in Alexandria, a city founded by Alexander in 331.

c. 300. Herophilus of Chalcedon; famous anatomist of Alexandria, was probably the first to regard the brain as the central organ of the nervous system. He carried out human dissections in public. His younger contemporary, Erasistratus of Chios, recognized the function of muscles. He believed that disease was caused by an excess of blood (plethora) in one or other locality of the body. He tried to explain respiration in terms of "Pneuma", an almost nonmaterial substance in air which was the cause of life.

c. 300. Erasistratus of Chios emphasizes the dangers of opium.

168. Revolt of Judas Maccabaeus against the Seleucids.

146. Conquest of Greece by Rome; destruction of Carthage.

70–19. Virgil.

63. Pompey extends the Roman boundaries to the Euphrates.

58. Julius Caesar makes the Rhine the Roman frontier in the North.

55 and 54. Caesar in Britain.

44. Caesar murdered.

B.C.

31. Octavian (Augustus) defeats the fleets of Antony and Cleopatra at Actium. Their suicide brought the end of Ptolemaic rule to Egypt, which became a Roman province.

4. Death of Herod the Great.

A.D.

26–36. Pontius Pilate, Procurator of Judea.

23–79. Caius Plinius Secundus (Pliny the Elder). A prolific author, he wrote, among other works, his famous *Historia Naturalis*, which was dedicated to Titus in A.D. 77 and was largely issued posthumously; it was translated into English in 1601. Pliny died in the eruption of Mount Somma (the lower summit of Vesuvius) which overwhelmed Pompeii, Herculaneum and Stabbia, and which is described by his nephew, Pliny the Younger.

The *Historia Naturalis*, in 37 books, contains a vast amount of miscellaneous knowledge, but is entirely indiscriminating; Pliny wrote down everything that came to hand, from old wives' tales to personal observations. His account of the mandrake mentions its use for diseases of the eyes, and describes, inadequately, the two forms of the plant (male and female). "Persons when about to gather the plant, take every precaution not to have the wind blowing in their faces; and, after tracing three circles round it with a sword, turn towards the West and dig it up." Directions are given for the extraction of the juice, which strikes some people dumb. A dangerous poison, the juice can be used as a narcotic before incisions are made in the body. It can also be used for snakebites and as an emetic. Notice is also taken of the soporific action of opium, but Pliny does not approve of its use.

37–*c.* 100. Flavius Josephus, Jewish statesman and soldier. He wrote the *Antiquities of the Jews* and the *Wars of the Jews*. It is in the latter (Bk. 7, Chap. 6, Sec. 3) that he describes the plant Baaras, which has strong affinities to the mandrake but is without soporific action. The method of obtaining the plant by means of a dog, later the standard procedure in mandrake catching, is here described for the first time.

c. 30–40. Issue of the *De Re Medicina*, by Celsus. This was the first classical medical work to be printed (1478), probably on account of the purity of its Latin. Celsus was not a physician and this book is all that remains of an encyclopaedia of natural science. Mention is made of the soporific qualities of poppy (Bk. 2, Chap. 32); mandrake is recommended as an ingredient in an eye ointment (Bk. 6, Chap. 6).

55–117. Tacitus.

64. Traditional date of the martyrdom of Saint Peter and Saint Paul.

68. Death of Nero.

c. 100. Pedanius or Pedacius Dioscorides of Anazarba in Cilicia issues his *Herbal*, which was to remain the chief authority on pharmacy until the end of the 16th century. There is some doubt about his date, but there seems to be no reason for believing that he was a surgeon in the army of Nero. His book leans heavily on Pliny and on other early writers. His accounts of the poppy and the mandrake are obviously drawn from the *Historia Naturalis*. He was, however, a physician, and is apparently the first medical man to describe the soporific effect of mandragora. His use of the word "anaesthesia" has already been mentioned.

A.D.

117–138. Hadrian.

c. 130–200. Galen of Pergamum. The system of medicine and physiology which Galen formulated remained authoritative until Vesalius and other anatomists in the 16th, and William Harvey in the 17th century, exposed the fallacies of his ideas. His more than 100 works, all dogmatic and plausible, played their part in preventing further scientific investigation for 1,300 years. He recommended a paste containing opium as an application for toothache; opium has no local analgesic action.

D. *The Age of Superstition* (A.D. 200–1454)

This period, from the death of Galen to the invention of movable type by Gutenberg, is contemporaneous with the decline and fall of the Roman Empire: Constantinople fell to the Turks in 1453. With the death of Galen Graeco-Roman medical science may be said to have come to an abrupt end. What medical authors there were contented themselves with writing commentaries on the works of Galen, which were kept alive, at least in Byzantium (Constantinople) after the transference of the seat of government thither from Rome in A.D. 330. However, the anarchical state of the rest of Europe led to a rapid decline in learning, and the Greek texts could no longer be read outside the domains of the Eastern Roman Empire. Grossly distorted translations of excerpts from such classical authors as Aristotle and Galen passed current for true learning, while much ingenuity was wasted in syllogistic quibbles about these texts. Observation was no longer used: what the ancients had written was true, and, if observation differed, observation was wrong. For instance, anatomical dissection ceased and was replaced by reading the Galenic texts, although Galen himself had probably never dissected a human being. This is the period of scholastic authoritarianism.

Three other forces also militated against science at this time: Religion, Astrology and Alchemy. The spread of Christianity in Europe with its answers to all problems, spiritual as well as temporal, disposed men to think of the world to come rather than the world in being. Furthermore, the accepted implication of religion was that pain and disease, like famine or poverty, were punishments prescribed by God for wickedness, either of the sufferer or of mankind at large. There was, therefore, something sacrilegious in attempting to fight disease or to prevent pain. It must not be forgotten, however, that in Western Europe at least, the Church was the sole repository of learning, the preservation of which was later to bear fruit. Until after the Reformation the Church had a vested interest in fostering antipathy to science, and she virtually forbade all experimental investigation; thus it was only at the risk of a charge of heresy that the doctrines of Aristotle could be challenged. This had a close bearing on the second factor, Astrology, for the close interdependence between Microcosm (Man) and Macrocosm (the Universe) which Aristotle had postulated came to be interpreted as meaning that worldly events could be foretold by the movements of the heavenly bodies, and there are certain passages in the Bible which will support such a construction. Such belief could not but be fatal to scientific enquiry.

Alchemy, the theoretical basis of which was laid down towards the end of the first millennium in the Moslem Empire, spread to Europe, partly as a result of the Crusades. Being a scientific investigation, it came into conflict with the Church, and, being based on false premises, weakened the cause of science still further.

The establishment of Christianity as the Roman official religion was soon followed by the growth of heresies: many of the heretics migrated rather than conform, and thus the Nestorians (Monophysites) carried into Asia Minor and the Near East the works of Galen in Greek. Later came the flood of Moslem conquest, followed in due course by the great period of Moslem culture. Galen's works were translated into Arabic, and commentaries were written on them by such men as Rhazes and Avicenna. These translations were infinitely better than anything available in Western Europe and were gratefully accepted when they began to appear in Latin, a further translation which was facilitated by contacts with the Moslem Empire, not only by the Crusades, but also in Spain, the southern two-thirds of which was Moorish, and in Sicily. The improved texts of Galen stimulated learning but not observation, for scholastic authoritarianism was still the ruling force and was to remain so until the Renaissance. However, a greater appreciation of what Galen and others had actually written became apparent, and schools of learning (universities) began to be founded, first in Italy and then elsewhere.

From this it can be seen that there was little possibility of an extension of medical knowledge during this period of twelve and a half centuries; indeed, there had been a diminution of it through over-emphasis on the wrong things. Furthermore, the inadequacy of the description of plants led to obvious difficulties, the more so as *Herbals* written, for example, in Asia Minor, were used in France and elsewhere where the flora was very different. The systematic identification of plants was, of course, not yet conceivable.

MANDRAGORA

We have seen the first reference to mandrakes and mandrake-like plants in Homer and the Bible. References multiply as time passes and are beginning to appear in medical works at the end of the scientific period, usually copied from Pliny, whose "authority", although he was not a medical man, was great. The adoption of the mandrake in medicine, chiefly as a soporific, was parallel to its use socially in magic. That no such plant exists, or ever did exist, in no way interfered with this. The finding of any anthropomorphic root blended well with earlier tales, and fantastic stories about the dangers and difficulties of obtaining specimens explained the absence of supplies. Later the name atropa mandragora was to be given to a plant of the family of Solanaceae, and this added to the confusion of thought about mandrakes.

As we have seen, primitive man naturally turns to magic in his attempts to master disease, and sympathetic magic, in which, for instance, a stone like a tooth can cure dental disease, is a method

which has had its appeal in all ages. The outstanding feature of the mandrake is its anthropomorphism, and this human shape led automatically to belief in its magical properties. Andrew Lang has summed up well the medieval ideas about this impossible root in *Custom and Myth* (1893): "He who desires to possess a mandrake must stop his ears with wax, so that he may not hear the deadly yells which the plant utters as it is being dragged from the earth. Then, before sunrise on a Friday, the amateur goes out with a dog, 'all black', makes three crosses round the mandrake, loosens the soil about the root, ties the root to the dog's tail and offers the beast a piece of bread. The dog runs at the bread, drags out the mandrake root, and falls dead, killed by the horrible yell of the plant. The root is now taken up, washed in wine, wrapped in silk, laid in a casket, bathed every Friday, and clothed in a little new white smock every new moon. The mandrake acts, if thus considerately treated, as a kind of familiar spirit, 'Every piece of coin put to her overnight is found double in the morning'."

All this is similar to belief in the healing power of the unicorn's horn, and strange tales about impossible animals and the methods of catching them, which passed for truth in these credulous days. Belief in magic was, of course, universal; the mandrake was especially magical in nature, and its use in medicine was merely one instance out of many in which magic and superstition were introduced into therapeutics. (For further evidence see the entry "1431. Execution of Joan of Arc" below.)

Very occasionally rational attempts at pain relief were employed in the "Age of Superstition", but these events failed to influence mankind and played no part in the discovery of anaesthesia. The statements by John Arderne, Theodoric and others must not be taken at their face value. The methods which they describe were merely amplifications of the tales by Pliny and Dioscorides, and depend on "authority", not on personal observation. In this regard, Arderne's *Treatise of Fistula in Ano, etc.*, although describing a method of pain relief (which would not have worked), also describes how his patients were restrained at operation.

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c. 220–230. Hua-T'o, Chinese surgeon. According to the *Kou-kin-i-tong*, he used a preparation called "mayo" in order to induce surgical anaesthesia. Unfortunately the *Kou-kin-i-tong* is dated some thirteen or fourteen centuries after Hua-T'o's death, and cannot therefore be relied upon as evidence.

c. 250. Approximate date of the *De Animalium Natura* of Claudius Aelianus, in which Josephus's story of using a dog to uproot the magical plant "Baaras" is first applied to the mandrake.

296–373. St. Athanasius. The date of his birth is doubtful, and may have been as late as 326. The creed which bears his name is probably 5th century.

303. Martyrdom of Saint Cosmas and Saint Damian, later the patron saints of Medicine and Pharmacy, in the persecution of Diocletian. They were held in special honour by the Confraternity of Surgeons in Paris and are remembered in the name of the College of St. Côme.

318. Arian Controversy begins.

325. The first great church council, held at Nicaea.

330. Constantine removes the seat of government from Rome to Byzantium, renamed Constantinople.

335. Closing of the temples in Constantinople by order of Constantine. Among the temples closed were, of course, the Asklepieia.

354–430. St. Augustine. He was Bishop of Hippo, in North Africa, from 395 until his death.

361–363. Julian the Apostate, Emperor of Byzantium, tries to re-establish the pagan religion.

c. 400. Apuleius (Lucius Apuleius Barbarus) prepared a *Herbal*, mainly by copying Pliny or Dioscorides. The mandrake receives attention in a manner which remains fairly constant in future medical works. The "dog story" is copied from Aelianus. The *Herbal* of Apuleius was frequently re-issued and translated into many languages.

410. Alaric the Visi-Goth sacks Rome.

431. Deposition of Nestorius, Patriarch of Constantinople, starts the Nestorian schism. The Nestorians carried the Greek classical works to Asia, where they were later translated into Arabic, and filtered back into Europe, as described above.

449. The Saxons settle in Britain.

452. Attila the Hun.

493. Theodoric the Ostro-Goth tries to establish a civilized kingdom in Italy.

c. 502–565. Aëtius of Amida, the first Christian physician of note. Mandrake is mentioned in his *Tetrabiblion*.

c. 570–636. Isidorus, Bishop of Seville. He mentions the pre-operative use of mandragora wine.

622. Muhammad (born 571) flees from Mecca to Medina (the Hajira, or flight, from which the Moslem calendar is dated). Since the Moslem calendar is lunar, not solar, conversion of dates to the European calendars is not easy.

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625–690. Paul of Aegina, the last Byzantine surgeon of note.

634. St. Aidan.

644. Moslem conquest of Egypt.

661. The Umayyad Caliphate begins; Damascus now the capital of the Arab world.

672–735. The Venerable Bede, first English historian.

705–718. The Moslems conquer North Africa, Sicily and Spain.

732. Charles Martel defeats the Arabs at the Battle of Tours.

750. The 'Abbasid Caliphate begins; Baghdad becomes the capital of the Eastern Arab Empire.

756. Establishment of the Umayyad Western Caliphate at Cordova.

763–809. Harun ar-Rasheed. He succeeded to the Eastern Caliphate in 786 and established the golden age of Baghdad. Although many of the stories in "The Thousand and One Nights" refer to him, it is not to be supposed that the tales approach to this antiquity.

776. Jabir ibn Hayyan, whose name appears in modern anaesthetic books as Djafar Yeber and who was known to medieval scholars as Geberus, flourished in al-Kufah at about this date. He was the father of alchemy, and began the search for the magical substance which would transmute base metals into gold (the "Philosopher's Stone"). He is the reputed author of many treatises, some of which were translated into Latin. In one of these translations there is an interpolated passage, absent from the Arabic original, describing the manufacture of "oleum vitreoli" (sulphuric acid). This led to a belief at the end of the nineteenth century that he was the discoverer of "oleum vitreoli dulce" (sulphuric ether).

800. Charlemagne, King of the Franks, rules from the Elbe to the Ebro.

850–923. Abu-Bakr Muhammad ibn Zakariya, known from his place of birth as Rhazes, was probably the greatest and most original of all the Moslem physicians and one of the most prolific as an author. He was the first to distinguish between smallpox and measles.

c. 850. Compilation of the Bamburg *Antidotarium*, which contains an account of the "spongia somnifera", made by steeping a sponge in a decoction of many herbs, including mandrake. Inhalation of the fumes from the sponge were said to produce unconsciousness. A similar recipe is found in the Codex of the Abbey of Monte Cassino, which had been founded by St. Benedict in 529. Since the drugs used were not volatile, the prescription could have been of little use: perhaps it was never used.

855. The Northmen begin to settle in France and England.

871–901. Alfred the Great.

962. Otho the German becomes Roman Emperor of the West.

980–1037. Ibn Sina (Avicenna), the most illustrious name in Arabic medical annals after Rhazes. His *Canon of Medicine* was mainly a recapitulation of Galenic writings and was in great demand up to the time of the Renaissance. He repeats the usual nonsense about mandragora, omitting the dog. However, he thought poppy extract a better sedative.

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994. Death of 'Ali ibn al-'Abbas (Haly Abbas), author of *al-kitab al-Maliki* (*Liber Regius*) and *Thesaurus of Medicine*. His greatest contribution to medicine was a proof that, in the act of parturition, the child does not come out by its own efforts but is pushed out by the muscular contractions of the uterus.

1001. Moslem conquest of Northern Hindustan.

c. 1050. Compilation of the Saxon Leech book, known as the *Lacnunga*. Amongst a great deal of magic are a few rational notions, one of which is that chilling a part by the application of cold water deadens the pain of opening an abscess. This seems to be the first recorded use of refrigeration analgesia.

c. 1050. Abu al-Qasim (Albucasis), greatest surgeon of the Western Caliphate, writes his textbook, *The Method*, which was to become the standard work on surgery.

1066. William the Conqueror defeats Harold at the Battle of Hastings.

1095. Peter the Hermit preaches the Crusade.

1098–1179. "St." Hildegarde of Bingen. In her book, *De Plantis*, she describes the mandrake which, being "formed of the moistened earth wherewith Adam was created," has magical properties.

1099. The capture of Jerusalem by the Crusaders.

1126–1198. Ibn Rushd (Averroës), great physician of the Western Caliphate, did much to aid the spread of Aristotelian ideas from Arabia to Europe. He knew that smallpox conferred immunity from future attacks. His work as a physician was eclipsed by his reputation as a philosopher.

1135–1204. Mosheh ben Maimon (Maimonides), court physician to Salahud-Din (Saladin) and preeminent among Jewish medical men of all ages. Like ibn Rushd, he was even more respected as a philosopher than as a physician.

1137. Foundation of St. Bartholomew's Hospital by Rahere.

c. 1140. Nicolaus Salernitanus revives the "spongia somnifera" in his *Antidotarium*.

1162. The first use of heraldry in England (Great Seal of Henry II).

1170. The murder of Thomas à Becket at the altar in Canterbury Cathedral.

1187. Jerusalem falls to the Moslems. The diminishing Frankish kingdom survives for a further century.

1200. Charter delivered to the University of Oxford.

1205–1296. Theodoric Borgognoni; his *Cyrurgia* describes a method of surgery which approaches to the aseptic. His description of the spongia somnifera is very well known and is quoted in all histories of anaesthesia. Ugone di Lucca (Hugo of Lucca), his teacher and, perhaps, father, died in 1252. The

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description of the "mandrake broth" is presumably derived from the *Antidotarium* of Nicholas Salernitanus.

1214–1294. Roger Bacon.

1214. At the Battle of Bouvines King John loses all his French possessions.

1215. Magna Carta sealed by King John at Runnymede.

1216. St. Francis of Assisi and St. Dominic found orders of friars; they meet.

1227–1274. St. Thomas Aquinas.

1234–1315. Raymond Lull or Lully; he is said to have described a substance which he called "oleum vitreoli dulce", but Keys could find no evidence of this on searching his works.

1265–1321. Dante.

1276–1326. Mondino; became professor of anatomy at Bologna in 1306. He carried out his first public dissection of the human body in 1315, and this occasion marks the revival of interest in anatomy.

1285. Armati (?) of Florence, died 1317, invents spectacles.

1295. First sitting of the complete English parliament.

c. 1298–1368. Guy de Chauliac. He was probably the greatest of the medieval European surgeons. His *Chirurgia Magna*, first issued in 1363, continued to be much used until the end of the 16th century; in it he mentions the "spongia somnifera" of Theodoric but does not indicate that he himself advocates its use. He disapproves of the pre-operative use of opium, since some who have had it have gone mad and consequently died.

1307–*c.* 1390. John Arderne. From about the time of the Black Death, when his wife died, he practised at Newark, but removed to London in 1370. He issued two important treatises, that of "Fistula in Ano, Haemorrhoids and Clysters" being issued in 1376. In it there is a description of an ointment compounded of mandrake and many other herbs for producing surgical anaesthesia. Needless to say, such a prescription would have been valueless. D'Arcy Power (*De Arte Phisicali et de Cirurgia*, 1922, p. viii) writes: "His medical treatment was essentially that of the Saxon leeches, treatment by spells, herbs and nasty or innocuous substances. In such matters he had no critical faculty but believed what he was told regardless of its source." This, of course, is typical of the medieval period, and accounts for all the mandragora broths, ointments and sponges mentioned by

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various authors. As a surgeon he was of great repute and the treatise above mentioned describes, for the first time, the operation for fistula in ano still in use today.

1309. Removal of the Papal seat to Avignon.

1314. The Battle of Bannockburn.

1315. The Swiss secure their independence, a factor of great importance in the later establishment of printing presses in Basle.

1328–1400. Chaucer. His works contain several references to narcotic drugs at the level which might be expected.

1340. Edward III claims the French crown.

1346. The Battle of Crécy; the first use of guns in war.

1349. The Black Death ravages England, having spread across Europe in the previous two years. In addition to the important social effects, this plague opened men's eyes to the importance of *infection* as a cause of disease and led to the development of quarantine stations at strategic points. This was perhaps the greatest step forward taken in this period.

1364. Formation of the Hanseatic League.

1411. Foundation of the University of St. Andrews.

1415. Battle of Agincourt.

1431. Execution of Joan of Arc. Her trial for witchcraft and heresy included a series of examinations, at one of which she was accused of possessing a mandrake. She denied this, and said that she knew mandrakes were dangerous and evil things to keep. The important point is that both she and her assessors knew that mandrakes were evil and magical. At about this time appeared an engraving by Jaspar Isaacs showing witches preparing for their Sabbat: a mandrake is shown in the foreground. Later about 1600, Franken used this engraving as the basis of his painting of a similar subject; he again depicts the mandrake.

1453. Constantinople falls to the Turks.

1454. Gutenberg prints from movable type. Printing was known to the Chinese many centuries earlier, but the discovery was stultified by the multiplicity of "characters". Gutenberg's discovery depended for its acceptance on the previous introduction of papermaking into Europe, which seems to have begun in Italy, about 1270. The knowledge of papermaking was carried by the Moslems from China to Europe, and paper was manufactured in Moorish Spain for some considerable time before the craft spread to Christian Europe.

(*To be continued*)

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SYMPOSIUM ON ANAESTHETIC TEACHING

On September 27 and 28, 1956, consultants from all parts of the British Isles, as well as from abroad, attended a symposium on anaesthetic teaching. This was organized by the University of London at the Postgraduate Medical School.

Dr. John Gillies presided on the first day, when the symposium, which had been introduced by Professor Ian Aird, was opened by Professor W. D. M. Paton, who dealt with postgraduate teaching in pharmacology. The main problem was how to combine the teaching of the rational basis of therapeutics with bringing students up to date on the ever increasing stream of important new drugs in the limited time available. Additional problems arose from the interest taken in examination questions, and from students' pre-occupation with the latest "fashion" in therapeutics. To teach so that a man's therapeutic practice would be successful, rational, and interesting to himself would depend increasingly on collaboration between preclinical and clinical teachers. He stressed a need for more intercommunication between pharmacologists and anaesthetists, which could be nurtured by mutually repeated visits to the operating theatre and the laboratory.

Professor J. McMichael touched on how postgraduates should be instructed in simple methods of physical examination and stressed the greater value of the history. He indicated a need for physicians and anaesthetists to combine in the investigation of the causes of disorders following surgical operations.

Professor W. W. Mushin detailed the "Aims in Training the Specialist Anaesthetist". All specialists should have a wide knowledge of medicine and of the basic sciences and be possessed of a mature personality. The anaesthetic specialist must, in addition, attain a high degree of technical proficiency, and a knowledge of how to apply basic science to his own subject and in other fields. He must be temperamentally fitted for team work, and should have a sympathetic approach to patients. The possession of all these as well as a critical outlook would qualify him to advise his hospital in clinical and other matters.

Professor Mushin outlined the special needs and difficulties of the trainee anaesthetist, and emphasized that only in the teaching hospital or in the largest nonteaching hospitals can these needs be adequately met. The smaller peripheral hospital was unlikely to have the facilities either clinical or scientific to provide a proper background for training the future specialist anaesthetist.

Points raised in the discussion were that: (1) trainees were being used as "a pair of hands"; (2) the exchange system between the teaching hospital and the periphery was difficult because of the lack of suitable accommodation; (3) the primary examination for the F.F.A.R.C.S. diploma was a difficulty because at this stage the graduate was mainly at work in the clinical field; if adequate knowledge could be gained at the undergraduate stage, the primary examination might become unnecessary; (4) the teaching hospital contact should be during the first year and at the time of the senior registrarship.

Drs. E. J. D'Arcy and E. Harvey Franks put the case for the trainee. Dr. D'Arcy felt the need for a closer relationship between consultants and trainees and for avoiding questions remote from anaesthesia in the examination. Dr. Franks drew attention to the disproportion in extent of practical opportunities existing at teaching, nonteaching and service hospitals, the former offering the greatest scope and the latter the least. It would seem desirable to have more exchanges between the civil and service hospital than exists at present but, as the discussion showed, the service needs had to be considered.

Dr. G. W. Organe presided on the second day when Dr. R. F. Woolmer opened with a paper on "Training in Methods of Research". He doubted whether it was worth attempting to give

all doctors training in methods of research, and he was not in favour of the extensive programmes as carried out in some quarters abroad. He thought, however, that more could be done within the present curriculum to interest students in research, and he favoured Professor Paton's approach to this.

Dr. F. Holmes spoke on "Some Special Problems in the Teaching of Obstetrical Anaesthesia", and discussed the relative merits of local and general anaesthesia in obstetrics. He was in favour of local methods whenever general anaesthesia presented special dangers to the foetus.

He used d-tubocurarine-chloride and light general anaesthesia after gastric aspiration with a polythene tube. He avoided both endotracheal anaesthesia and controlled respiration.

A lively discussion followed during which several speakers pointed to the value of teaching medical students how to perform pudendal block with a view to employing this method in domiciliary midwifery; others felt that this procedure might endanger the patient because of sepsis. One suggestion was that the general practitioner ought to be utilized to train students in chloroform anaesthesia. Nitrous oxide oxygen and chloroform was another possibility but there were difficulties in arranging sessions with general practitioners.

Dr. A. A. Mason spoke on the teaching of hypnosis, indicating how this procedure could be carried out by anaesthetists in their daily work.

Dr. T. C. Gray drew attention to a fundamental change in outlook in undergraduate teaching. The aim should now be to produce a graduate who could adapt himself for any branch of medicine. The head of each specialty would have to decide how best he could contribute to the more generalized course. As the law stands today the medical graduate is expected to be able to give an anaesthetic to any patient. Dr. Gray emphasized the need for reform. But until such an event, undergraduate teaching commitments must remain despite the lessened opportunities. He outlined the undergraduate course in practical anaesthesia at Liverpool University and emphasized the importance of a personal interview at the end of the course, so that where experience was lacking the deficiency could be remedied.

Professor E. A. Pask discussed what should be taught to undergraduates. The problem was how to reduce the extent of the teaching material and yet give more emphasis to things of value to all doctors. He thought that the opportunity for the student to "do something with his hands" with responsibility was valuable and made the point that the modern tendency to use instruments of various kinds is demanding practical abilities. The anaesthetic course had much of practical value to give in a general medical course, particularly in applied pharmacology and physiology.

A Handbook of Medical Hypnosis. By Gordon Ambrose and George Newbold. Published by Baillière, Tindall and Cox. Pp. 255. Price 21*s*.

It might have seemed more appropriate for this book to be reviewed by a psychiatrist, but there seems much to be said for recording the impression it creates on one who is far from a specialist in this subject but who is aware of its apparently unexplored potentialities. The book is described as an introduction for practitioners and students, and in the opinion of the reviewer it fulfils its purpose well. After a chapter on the history of medical hypnotism there is a simple review of the phenomena of the hypnotic state and of the methods for attaining it. So far all is well, but it is doubtful whether the ensuing chapters on the treatment of various conditions in general medicine and of neuroses should come within the scope of the general practitioner without the guidance of the expert. It does seem that the amplification of this technique and the treatment of psychiatric disorders requires greater consideration and experience than could be obtained from so casual an acquaintance with the subject. There is only a brief review of the use of hypnotism in surgery and dental surgery (4 pages), but a fuller description is given of its importance in obstetrics.

It is obvious that every anaesthetist uses suggestion in his daily practice and there is no doubt that many produce a stage of hypnosis during a gaseous induction of anaesthesia. It would seem that further extension of this technique into our field, despite some recent enthusiasm, is not likely to be practicable on a large scale. What will be of the greatest interest to all who are aware of the trend in recent years to re-emphasize the psychosomatic aspect of medicine and the body's reaction to trauma will be the description of the use of hypnotism in the treatment of conditions such as chronic dyspepsia, ulcerative colitis and various dermatological afflictions.

This is a book which will be found to be an interesting introduction to what Dr. William Moodie in his preface calls, ". . . not only a very valuable therapeutic weapon, but a fascinating and enlightening field of study . . ."

T. Cecil Gray

The Medical Annual, 1956. Edited by Sir Henry Tidy and R. Milnes Walker. Published by John Wright and Sons Ltd. Price 37*s*. 6*d*.

The 1956 edition is slightly smaller than its predecessor and only eight pages of it are devoted to anaesthesia and analgesia, so even the busiest of anaesthetists can find time to read them all and find their task rendered easier by the presence of seven illustrations. The practitioner's index at the end of the book will be found useful for reference, but neither in it nor in Dr. Hewer's article is made any mention of the new inhalation anaesthetic Fluothane. That may, of course, be due to the fact that it is too new, Johnstone's account of the first 500 cases appearing as recently as in the September number of the *British Journal of Anaesthesia*.

E. Falkner Hill

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